"*I would have everie man write what he knowes and no more.*"—MONTAIGNE

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EDITORIAL

ANAESTHESIA AND THE N.H.S.

This month marks the eighth anniversary of the institution of the National Health Service. Probably all will agree that the fears and apocalyptic forebodings expressed by many during the period of negotiation have not really been justified. Blemishes in the structure of the edifice and dissatisfactions among its inhabitants there undoubtedly are, but these are attributable more to the Treasury rather than to any fundamental defect in the service. For the most part—there have been and are exceptions—wisdom in administration and in the interpretation of regulations has allayed the worst fears of bureaucratic interference.

Certainly the realist cannot help but realize that as far as the hospital services are concerned under present conditions of prices and costs the voluntary system could not have survived. Modern conditions of specialist practice and the cost of up-to-date equipment would have been crushing burdens to the old regime. A form of subsidization to an extent which implied the adoption of full responsibility for the hospitals by the State had to come, and the medical profession has done well indeed to maintain that degree of autonomy and independence which is essential for the wellbeing of the patients—individuals after all comprise the State.

It must be true to say that the N.H.S. has provided the sick of this country with the best anaesthetic service in the world. There is plenty of room for improvement still, but nevertheless it is a great deal that every hospital has highly trained and qualified consultant staff and that it is generally agreed that where major surgery is undertaken consultant anaesthetic cover should be provided.

Before the N.H.S. the possibility of an anaesthetist making a reasonable income from his specialty alone was remote except for a comparative few working in large centres. The result was poor recruitment and slow advance. The financial stability of the new system has altered this and anaesthesia has developed into an attractive vocation assuring at least an adequate income and work which in many of its aspects is more scientific than any other branch of clinical medicine.

Financial stability has permitted the Faculty to demand a period of training and a standard of academic achievement which enables those awarded its Fellowship to hold their heads high in any clinical company.

Equal status implies equal responsibility. It is alleged, we hope without foundation, that unfortunately there are a few who in hospital cut their services to the minimum, and others who feel unable to assume their full share in committee and administrative work. The latter is generally less attractive to medical men than the more vocational and interesting work in hospital or laboratory. It is of course true that many anaesthetists spend too many hours under the trying and demanding conditions of operating theatre work, so that they can hardly be blamed for feeling disinclined to assume other responsibilities. Perhaps this should be remembered by critical consultants in other fields of medicine whose routine work is less demanding. If, however, there are those who are not playing the game either in hospital or out of it, we hope that they will not resent us pointing out that the good of the whole specialty, its influence, prestige, and perhaps indirectly its monetary awards, will be affected by their attitude.

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ANAESTHESIA WITH CONTROLLED POSITIVE AND NEGATIVE PRESSURE RESPIRATION

PART I: A CLINICAL EVALUATION

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CONTINUOUS but slow progress is being made towards solving the numerous problems arising from the attempt to maintain a near-normal physiological state in patients under general anaesthesia. The depressant effect of drugs used in preoperative medication and in general anaesthesia on respiration and on circulation have been amply demonstrated (Schmidt and Harer, 1923; Hershey and Zweifach, 1950; Cohen and Beecher, 1951; Johnson, 1951; Holaday et al., 1952; Johnstone, 1955; Eckenhoff, et al., 1955). The means for avoiding this depression are still under intensive scrutiny and form the subject of this review.

The value to pulmonary ventilation of assisted or controlled breathing, in an attempt to avoid to some degree the acid-base disturbances when the pleural cavity remains intact, has been substantially accepted by most clinicians since Watrous et al. reviewed this subject in 1950-51. The possible depression or elevation of blood pressure and the hazards of respiratory acidosis under these circumstances have also been demonstrated (Dripps, 1947). These pathological changes are further aggravated when the pleura is open, and their prevention has been studied intensively and carefully reviewed during the past five years (Beecher, 1951; Dobkin and Van Bergen, 1952; Buckley et al., 1953; Mushin and Rendell-Baker, 1953; Papper and Ngai, 1954; Dripps and Severinghaus, 1955). Premedication with ataractic drugs (such as chlorpromazine), reduction or complete elimination of opiates in premedication, use of light levels of anaesthesia, judicious use of muscle relaxants and the use of balanced anaesthetic techniques have all assisted in reducing these defects (Dobkin and others, 1954, 1955, 1956; Little and Stephen, 1954). The major problem which exists at present is to determine whether the augmentation of breathing in the anaesthetized patient can be improved by

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using mechanical devices (Mautz, 1939; Maloney et al., 1952; Pask, 1955; Gibbon and Haupt, 1955); to recognize the physiological effects of such devices (Humphreys et al., 1938; Beecher et al., 1943; Bennett et al., 1944; Werko, 1947; Thompson and Rockey, 1947; Cournand et al., 1952; Bjurstedt et al., 1953; Price et al., 1954; Maloney and Hanford, 1954; Mørch and Benson, 1954; Saklad, 1954; Lucas and Milne, 1955); and to determine whether the patient can benefit further by the use of a period of subatmospheric pressure in the breathing cycle (Maloney et al., 1953; Hubay et al., 1954; Allbritten et al., 1954). This information is urgently required to improve the cardiovascular and respiratory management of patients undergoing intrathoracic operations.

MATERIALS AND METHODS

In order to investigate some of these problems clinically the major effort in this study was devoted to the evaluation of a fully automatic pressure respirator, supplied by the Ohio Chemical and Surgical Equipment Company of Madison, Wisconsin, U.S.A., in which positive, negative, and atmospheric pressures could be timed automatically and could be controlled by means of timed delay relay switches for duration of phases and rate of respiration. Indicators recorded phase pressures, and calibration of the ventilating bellows indicated tidal volume. The tidal volume of respiration was delivered through nondistensible (Tygon) tubing from the respirator to the rebreathing inlet of a Heidbrink circle absorber and through semidistensible tubing from the Heidbrink inhalation and exhalation valves to the endotracheal tube.

Recent studies with this experimental respirator in dogs have shown that with a closed chest intermittent positive-negative pressure respiration increases the venous return to the heart to a significant degree over that measured during positive-atmospheric pressure respiration, particularly when the animal is suffering from hypovolaemia (Brecher, 1952; Hubay et al., 1954). This would appear to be of physiological benefit since the mechanism of increased venous return to the heart corresponds to that observed with spontaneous respiration (Brecher, 1953). With the open chest these studies have shown that intermittent positive pressure inflation of the lung impedes venous return, but the interposition of a negative phase between the positive phases did not benefit the circulation significantly (Ankeney et al., 1954). The accentuation of detrimental effects on circulation by the lateral position have also been shown (Hubay et al., 1955).

The effect on the structure of the lung of both positive and negative intratracheal pressure respiration was studied in dogs also. It was found that intermittent positive pressure of up to 40 cm water over periods of 2-4 hours were well tolerated when the chest was not opened. However, when a surgical pneumothorax was produced, intermittent positive pressures of 40 cm of water caused severe damage owing to repeated overdistension of the lung parenchyma. When alternating positive and subatmospheric pressures were employed no significant changes were observed when the pressures did not fall below −5 cm of water. With the chest intact, greater subatmospheric pressures in the trachea caused more severe damage

than when surgical pneumothorax existed, presumably due to direct transmission of the pressure to the lung parenchyma. When the pneumothorax was produced, high subatmospheric tracheal pressures did not reach the lung parenchyma owing to complete collapse of the small bronchioles at lower pressures (the "trapping" phenomenon). The most favourable intratracheal pressures which permit effective pulmonary ventilation with minimal damage to the lung parenchyma of dogs were obtained when +15 to +20 cm of water were used (Waltz et al., 1954).

After the circulatory and morphological studies were completed in dogs, the controlled clinical evaluation of the mechanical respirator was carried out as follows:

Group I. Normal lungs, controlled respiration — hand squeeze and experimental respirator.

Sixteen patients for elective surgery with clinically normal lungs were studied. These patients underwent a variety of surgical procedures both with and without surgical pneumothorax. Anaesthetic technique varied slightly in these cases (see Table I). A Heidbrink circle system charged with fresh soda lime was connected both to the experimental respirator and to the patient by nondistensible tubing (Tygon). Data was collected for the effect on acid-base homeostasis using arterial blood samples from the brachial artery, collected anaerobically in syringes moistened with heparin and immediately cooled to 4°C. These were analysed as follows: The pH was determined by a glass electrode pH meter at 37°C. Total serum carbon dioxide content and oxygen content (volumes per cent) were determined by the method of Van Slyke and Neill (1924). The partial pressures of carbon dioxide (pCO_2) in the arterial blood were calculated from the Henderson-Hasselbach equation using the determinations of pH and total CO_2 (Van Slyke and Sendroy, 1928; Singer and Hastings, 1948).

Respiration data was collected as recommended by Radford (1953). Pulmonary ventilation (volume of respired gas) was measured with a Lilly pneumotachygraph (Lilly, 1950). Inspired gases passed between the walls of an ice-cooled calorimeter chamber to reduce condensation of moisture on the pneumotachygraph monel metal screen. Tracheal pressure measurements were carried out at the level of the endotracheal tube connector through a Statham strain gauge. The mean airway pressure was then calculated by integration of the area under the curve of endotracheal tube pressures. Arterial blood pressure was measured from the brachial artery through an indwelling needle via a Statham strain gauge. Carbon dioxide in volumes per cent in the expired gases was determined through a Liston-Becker infrared absorption CO_2 analyser (model 16) connected to the expiration side of the Y connector on the endotracheal tube. This apparatus was previously calibrated with known and checked concentrations of CO_2 (Collier et al., 1955). The CO_2 concentrations were converted to mm Hg. Patients undergoing intrathoracic procedures were monitored by the electrocardiograph (lead 2) using a cardiotachyscope—a procedure considered essential (Johnstone, 1955) in all such operations (fig. 1).

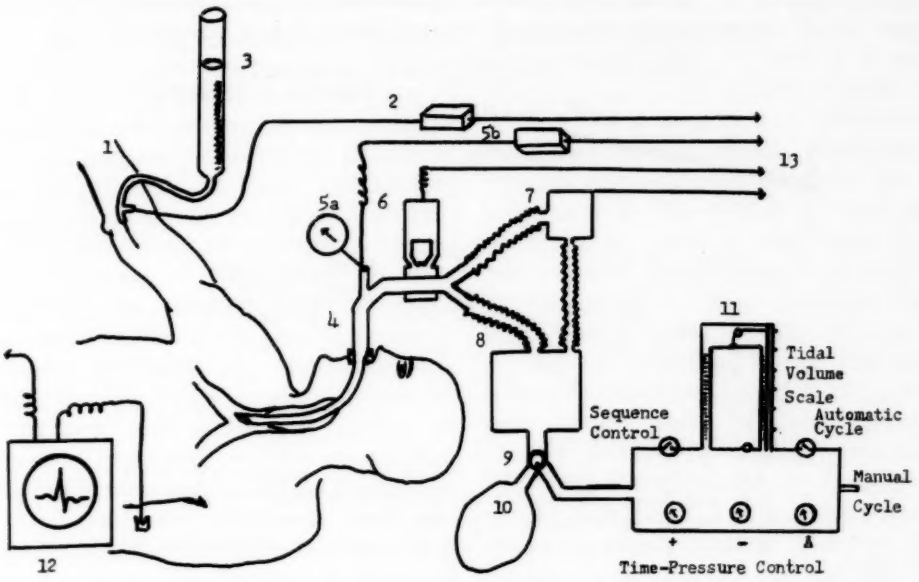

FIG. 1

Arrangement of anaesthetic and recording equipment for study of acid-base changes, and cardiorespiratory effects of controlled respiration.

(1) Cannula in brachial artery connected to 3-way stopcock with limbs for 0–250 lb/sq.in. Statham strain gauge to measure arterial blood pressure (2) and to draw arterial blood samples anaerobically or flush system with heparinized saline (3). Airway is controlled with a long-cuffed large-bore endotracheal tube connected to a curved catheter connector (4) with suction tube nipple, through which either Emerson aneroid positive–negative pressure gauge 5(a) or 0–0.2 lb/sq.in. Statham strain gauge (5b) is attached for measuring or recording airway pressure. A Lilly pneumotachygraph (6) to measure inspired and expired gas volume in anaesthetic circuit between catheter connector and Y connector. A Liston Becker infrared CO_2 analyser to measure carbon dioxide in the expired gases (7). Inspired gas is channelled through outer wall of a calorimeter chamber filled with ice water to prevent water condensation on pneumotachygraph screen (not shown in diagram) and expired gas passes through circle carbon dioxide absorber of a Heidbrink gas machine filled with fresh soda lime (8). A three-way stop-cock (9) is attached to the lower end of the soda lime canister to permit quick switch over from rebreathing bag (10) to the mechanical respirator (11). A cardiotachyscope to indicate the heart rate and the electrocardiogram (12). Blood pressure, airway pressure, gas exchange in the airway, and expired carbon dioxide are recorded on a 4-channel Sanborn Polyviso (13).

Group II. Abnormal lungs, controlled respiration with Blease pulmoflator.

Concurrently, a second study was carried out on 20 patients undergoing elective surgery for advanced pulmonary tuberculosis. All patients in this and the following study had diagnostic bronchoscopic examinations to ensure that no endobronchial disease existed. Anaesthetic technique was constant in all cases. Rate of respiration was fixed at 18 per minute, and no less than the minimum volume of respiration was provided as determined from the Radford nomogram corrected for anaesthetic factors (fig. 2; Radford et al., 1954; Radford, 1955). Pressures were set at +15 mm Hg for one-third of the respiratory cycle. The cardiotachyscope monitor

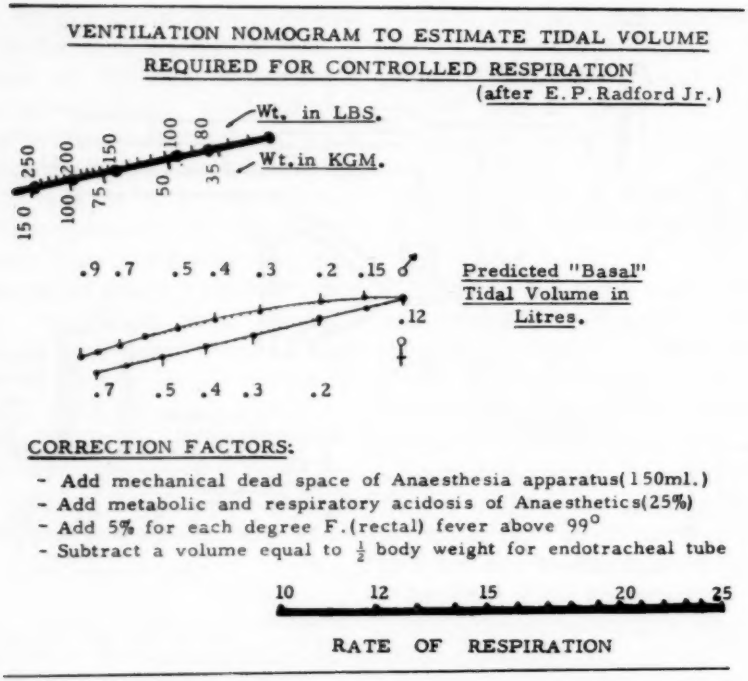
VENTILATION NOMOGRAM TO ESTIMATE TIDAL VOLUME
REQUIRED FOR CONTROLLED RESPIRATION
(after E.P.Radford Jr.)
Wt. in LBS.
Wt.in KGM.
150 250 100 200 75 150 50 100 35 80
.9 .7 .5 .4 .3 .2 .15
.7 .5 .4 .3 .2 .12
Predicted "Basal" Tidal Volume in Litres.
CORRECTION FACTORS:
- Add mechanical dead space of Anaesthesia apparatus(150ml.)
- Add metabolic and respiratory acidosis of Anaesthetics(25%)
- Add 5% for each degree F.(rectal) fever above 99°
- Subtract a volume equal to ½ body weight for endotracheal tube
10 12 15 20 25
RATE OF RESPIRATION

FIG. 2

was employed throughout the period these patients were in the operating room.

Patients in this group varied in age from 18 to 54 years and had relatively normal cardiac function. Pre-operative pulmonary function tests as recommended by Warring (1945), Wright and Michelson (1950), and Gaensler (1955) were carried out, including chest fluoroscopy for rib motion, diaphragm motion, and rate of filling and emptying of the lung; maximum breathing capacity, walking ventilation, total vital capacity, timed vital capacity at 1, 2 and 3 seconds and expiration time were recorded. In addition 11 of these 20 patients had bilateral bronchospirometry carried out employing a Carlens double lumen endobronchial catheter and double spirometer to determine the distribution of lung function. Anaesthesia was maintained with a Blease pulmoflator respirator (Musgrove, 1952) attached to the endotracheal tube by a to-and-fro carbon dioxide absorption system with semi-distensible tubing. The soda lime canister was refilled with fresh soda lime every hour. Airway pressures were checked on the anaesthetic machine and by an Emerson positive-negative aneroid pressure gauge at the endotracheal tube connector. Mean arterial blood pressure was measured in 14 of the 20 patients by means of an aneroid blood pressure manometer as described by Alman and Fazekas (1953).

Group III. Abnormal lungs, controlled respiration—hand squeeze and experimental respirator.

A third study was carried out on 73 patients for elective surgery with pulmonary changes due to infectious disease of the lungs. Most of these had varying degrees of pulmonary emphysema, fibrosis, venous-arterial shunts and gross defects in gas distribution. Sixty-nine of these patients were suffering from advanced pulmonary tuberculosis. During the maintenance of anaesthesia, respiration was controlled by hand squeeze during 45 operations, and a selected variety of pressure patterns on the mechanical respirator was used during 28 operations. The patients of this group had preoperative pulmonary function tests carried out as in Group II. Twenty-one of the 45 patients controlled by hand squeeze and 12 of the 28 patients controlled with the respirator had bronchospirometry studies carried out to determine the minute volume, oxygen consumption and vital capacity of each lung, as in Group II. In all patients of this group the basal tidal volume requirement was determined from the Radford nomogram corrected for anaesthesia factors as a guide to the minimum volume of respiration necessary for adequate alveolar ventilation. In the 28 patients managed with the experimental respirator, arterial blood samples were drawn prior to induction of the anaesthesia, 30 minutes after the patient was positioned and anaesthesia stabilized; 30 and 90 minutes after the pleura was opened; and again 180 minutes after the pleura was opened if the operation was prolonged. A final blood sample was drawn 30 minutes after the chest was closed. This timing was closely adhered to throughout, except where major vessels were about to be clamped or a major bronchus was to be opened. Syringes for arterial blood samples were prepared as recommended by Cournand. Four types of pressure patterns were employed on the respirator. *In the first*, +15 mm Hg/atmospheric pressure was used in 5 patients with phases timed 1:2 and rate 18/minute (the "ideal" pressure curve according to Cournand et al., 1952); *in the second*, +15 mm Hg/−5 mm Hg/atmospheric pressure was used in 8 patients with phases timed of equal duration (1:1:1) and rate 18/minute; *in the third*, +15 mm Hg/atmospheric pressure/−5 mm Hg was used in 7 patients with phases timed of equal duration and rate 18/minute; *in the fourth*, +15 mm Hg/−5 mm Hg was used in 8 patients with phases timed of equal duration and rate at 18/minute. As in the second study a cardiotachyscope monitor with lead 2 was employed throughout the investigation.

In none of these cases was it necessary to change the pressures in order to provide the basal tidal volume as determined from the Radford nomogram. Occasionally pressures were reduced to assist the surgeon during periods of difficulty in the dissection, and in others the pressure was periodically increased when expansion was thought to be inadequate.

Anaesthetic management for all intrathoracic cases in Groups II and III was as follows: the evening prior to operation the patient was sedated with 50–100 mg of chlorpromazine and 100 mg of quinalbarbitone by mouth, depending on the size and general physical state of the patient. The morning premedication con-

sisted of a deep intramuscular injection of chlorpromazine 25 mg and scopolamine 0.6 mg (for all patients) administered one hour prior to transport to the operating room. This preparation sedated the patients very well without grossly disturbing respiration or circulation (Dobkin et al., 1955). Upon arrival in the operating theatre the cardiotachyscope was attached and the patient's oropharynx was sprayed with 2–3 ml of 1 per cent amethocaine or 2 per cent lignocaine. This was followed by a preliminary period of breathing 100 per cent oxygen while a brachial artery cannula was inserted and two intravenous infusions with 15 gauge needles were started in an arm and leg vein. Induction of anaesthesia was carried out with a mixture of 500 mg thiopentone and 15 mg d-tubocurarine in 25 or 30 ml of distilled water (2 per cent or 1.8 per cent thiopentone). If the patient lay quietly during the preparations, the more dilute mixture was used. If the patient showed signs of restlessness during the preparations the stronger solution was used. The mixture was injected slowly until the patient was unconscious. The lungs were then inflated with 100 per cent oxygen employing artificial manual ventilation for 2 to 3 minutes by the clock. Endotracheal intubation was then performed with the largest long-cuffed endotracheal tube which would pass with ease or with a Carlens double lumen catheter. These were previously coated with 0.75 per cent cyclomethycaine jelly (Surfacaine (R)). Approximately 1 ml of the topical anaesthetic was sprayed through the endotracheal tube and the investigative equipment and anaesthetic circuit connected as shown in figure 1. Maintenance of anaesthesia consisted of nitrous oxide-oxygen inhalation in a 1:1 ratio using an intermittent closed circuit and controlled respiration with a Blease pulmoflator (Group II) or with a Heidbrink circle system (Group III) charged with fresh soda lime.

Ninety-six of the 99 patients for open thoracic surgery in these studies were operated upon in the lateral position, fully recognizing the fact that this is the worst posture from a pulmonary ventilation point of view (Beecher and Murphy, 1950; Barach and Beck, 1954). Three were "very wet" cases and were operated upon in the prone position on an Overholt table. As soon as the anaesthetic and surgical preparations were completed, respiration was mechanically controlled by intermittent inflation and deflation pressures as indicated in the tabular data. Intravenous anaesthetic supplements were given as necessary in doses considered appropriate to the occasion. All anaesthetic procedures were carried out by the same anaesthetist (A.B.D.) and the majority of the operative procedures performed by the same resident surgeon (A.S.). All operations on patients with pulmonary tuberculosis were carried out in an air-conditioned operating room maintained at a temperature of 70°F. and a relative humidity of 55-60 per cent.

RESULTS

In the patients with normal cardiopulmonary function (Group I), and where the *pleura was not opened*, adequate oxygenation and carbon dioxide excretion was evident when manually controlled respiration was conscientiously carried out. Clinically there was little evidence of

TABLE I

Miscellaneous operations.

Pt.	Age	Sex	Color	Physical State	Operation	Premedication	Position	Anaesthetics Thiop.	dTc	N_2O	Controlled Respiration Method	Pattern mm Hg	Phase Time Ratio	Blood Trans.	Anaes. Time Min.	Pleura Open Min.
					Simple Mastectomy											
M1	77	F	W	2	M25	A.6	S	425	12	50%	HS	+10-15,0	1:2	500	130	
					Chole.-CBD											
M2	63	F	W	2	M50	A.4	S	800	24	50%	HS Ohio	+10-15,0 +15,-5,0	1:2 1:1:1	1000	360	
					Incisional Hernia											
M3	68	M	W	3	M50	A.5	S	1000	30	50%	HS Ohio Ohio	+10-15,0 +15,0 +15,-5,0	1:2 1:2	0	200	
					Chole. CBD											
M4	47	F	W	2	M75	S.6	S	1200	40	50%	HS Ohio Ohio	+10-15,0 +15,0 +15,-5,0	1:2 1:2 1:1:1	500	200	
					Cholecystectomy											
M5	61	F	B	2	M50	A.4	S	600	18	50%	HS Ohio Ohio	+10-15,0 +15,-5,0 +15,-5	1:2 1:1:1 1:2	0	180	
					Chole. CBD											
M6	22	F	W	1	M50 S50	S.6	S	600	18	50%	HS Ohio Ohio	+10-15,0 +15,0 +15,-5,0	1:2 1:2 1:1:1	500	250	
					Chole. App.											
M7	22	F	B	1	M50	A.6	S	900	25	50%	HS Ohio Ohio	+10-15,0 +15,0 +15,-5,0	1:2 1:2 1:1:1	0	220	
					Nephrectomy											
M8	65	M	W	3	M50	S.6	L	Ether - O_2			HS Ohio Ohio	+10-15,0 +15,0 +15,-5,0	1:2 1:2 1:1:1	0	155	
					Laminectomy											
M9	17	M	W	1	C25	S.6	P	750	27	50%	Jeff.	+15,-5	1:1	1000	300	
					Laminectomy											
M10	39	M	W	1	C25	S.6	P	450	21	50%	Jeff.	+15,-5	1:1	0	120	
					Explor. Thorac.											
M11	63	M	W	3	M50	A.4	L	575 Sc. 700	12	50%	HS Ohio Ohio	+10-15,0 +15,0 +15,-5,0	1:2 1:2 1:1:1	1000	300	165
					Explor. Thorac.											
M12	53	M	W	2	M75	A.6	L	900	42	50%	HS Ohio Ohio	+10-15,0 +15,-5,0 +15,0	1:2 1:1:1 1:2	500	180	90
					Oesophagectomy											
M13	78	M	W	3	M50	A.6	S-L	1000	30	50%	HS Ohio Ohio Ohio	+10-15,0 +15,0 +15,-5,0 +15,0,-5	1:2 1:2 1:1:1 1:1:1	1000	8 hrs.	240
					Oesophagectomy						HS Ohio	+10-15,0 +15,0	1:2 1:2			
M14	54	M	B	3	M75	A.4	S-L	1350 Sc. 800	34	50%	Ohio Ohio	+15,-5,0 +15,0,-5	1:1:1 1:1:1	2500	9½ hrs.	225
					Explor. Thorac.											
M15	60	F	W	3	C25	S.6	L	575	20	50%	Jeff.	+15,-5	1:1	500	190	55
					Explor. Thorac.											
M16	72	M	W	3	C25	S.6	L	500	27	50%	Jeff.	+15,-5	1:1	500	150	85

C=Chlorpromazine. M=Meperidine. A=Atropine. S=Scopolamine.
Jeff.=Jefferson Respirator.

cardiovascular disturbances with the balanced anaesthetic technique employed. Mechanical control of respiration also maintained a near-normal physiological state regardless of the pattern used for mechanical respiration and had the advantage of freeing the hands of the anaesthetist. The only patient where normal pH could not be maintained even with the respirator was the single case in which ether was used (M8). In the six patients where the *pleura was opened* (M11-M16) only one serious fall in pH occurred (M16). This followed surgical manipulation in the thoracic cavity causing intermittent disturbance of ventilation and circulation in a functionally normal lung. The disturbance rapidly corrected itself after manipulation was discontinued. The older of the two patients who underwent oesophagectomy (M13) showed a definite cardiovascular change when intermittent positive-atmospheric pressure respiration was used. In both cases (M13, M14), however, pH and pCO_2 remained at a slightly alkalotic level regardless of the respirator pattern (figures 4 and 5). Tables I and II contain the pertinent data for the patients in this group.

The patients with abnormal lungs (Group II and III) due to advanced pulmonary infection (and the older patients with tuberculosis who probably suffered from some degree of cor pulmonale also) who underwent pneumonectomy, lobectomy and segmental resections are tabulated according to the operative procedures in tables III, IV and V. Preoperative pulmonary function studies and arterial blood data for these patients are tabulated in tables VI, VII and VIII.

The patients in this group varied in age from 13 to 69 years and the operative procedures varied in duration from 135 to 450 minutes. In none of these patients was there clear-cut clinical evidence of serious cardiovascular depression due to the method or pattern of controlled respiration either while the pleura was open or closed. There were no deaths among these patients either in the operating room or within 5 days of the operation. Except for suspected or clinically evident cyanosis in the immediate postoperative period in several cases, the postanaesthetic course was relatively uncomplicated in this group. Cyanosis was due either to "diffusion anoxia" (Fink, 1955) or to marked reduction in functioning pulmonary tissue (Bjork and Engstrom, 1955) and was corrected with 100 per cent oxygen by BLB mask. Patchy, segmental or lobular atelectasis developed in some of these patients in the early postoperative period. These were treated with endobronchial suctioning through a bronchoscope and routine physiotherapeutic measures.

For *the anaesthetist* the respirator provided the decided advantage of freeing his hands. This permitted him to check vital signs, to administer blood and anaesthetic drugs, and to keep charts. During use of the respirator, however, it was frequently evident that there was loss of close contact with the patient's state of anaesthesia. For *the surgeon* the respirator was of no particular disadvantage while the surgical dissection was progressing smoothly. However, there were certain respirator patterns which were definitely a nuisance and a hindrance, especially when difficulty with the dissection was encountered.

Two patterns were particularly disturbing to the surgeon, namely, that employing

TABLE II

Arterial blood data from patients managed entirely by controlled respiration.

Patient	pCO_2 mm.Hg. pH O_2 vol.%	pCO_2 mm.Hg pH O_2 vol.%	pCO_2 mm.Hg pH O_2 vol.%	pCO_2 mm.Hg pH O_2 vol.%	pCO_2 mm.Hg pH O_2 vol.%
M1	18 7.65 19.2 H.S. 75" S	19 7.62 19.3 H.S. 90" S	24 7.52 19.3 H.S. 130" S		
M2	27 7.55 14.6 H.S. 115" S	25 7.57 15.2 R. 15,-5,0 30" S	25 7.56 15.8 H.S. 40" S		
M3	33 7.45 17.3 Before Anaesthesia	24 7.56 19.1 H.S. 50" S	22 7.59 19.8 R. 15,0 30" S	25 7.57 21.9 R. 15,-5,0 40" S	24 7.58 21.2 H.S. 40" S
M4	28 7.53 17.4 Before Anaesthesia	19 7.64 19.4 H.S. 30" S	17 7.64 26.9 R. 15,0 45" S	17 7.63 22.1 R. 15,-5,0 30" S	
M5	33 7.51 15.8 Before Anaesthesia	29 7.51 21.5 H.S. 40" S	30 7.48 19.5 R. 15,-5,0 30" S	25 7.53 19.4 R. 15,-5,0 60" S	26 7.51 19.4 H.S. 30" S
M6	32 7.42 16.8 Before Anaesthesia	29 7.45 18.4 H.S. 45" S	25 7.51 16.5 R. 15,-5,0 40" S	22 7.54 17.4 R. 15,0 35" S	
M7	39 7.35 18.6 Before Anaesthesia	35 7.36 20.6 H.S. 60" S	24 7.50 19.3 R. 15,0 30" S	33 7.33 19.6 R. 15,-5,0 40" S	41 7.27 20.2 H.S. 30" S
M8	35 7.39 17.0 Before Anaesthesia	59 7.19 18.2 H.S. 45" L	56 7.19 17.1 R. 15,0 20" L	55 7.20 16.7 R. 15,-5,0 20" L	
M9	40 7.30 18.7 Before Anaesthesia	24 7.45 23.5 R. 15,-5 30" P	34 7.30 19.0 R. 15,-5 210" P	37 7.32 18.9 S.R. 45" S	
M10	22 7.57 18.2 Before Anaesthesia	14 7.65 21.5 R. 15,-5 90" P			
M11	33 7.44 17.3 Before Anaesthesia	38 7.37 19.6 H.S. 60" L	34 7.40 20.6 H.S. P-O 40" L	35 7.43 20.3 R. 15,0 P-O 75" L H.S. 30" L →	46 7.28 19.9 R. 15,-5,0 P-O 135" L 44 7.31 18.6
M12	34 7.44 15.0 Before Anaesthesia	36 7.49 15.9 H.S. P-O 25" L	46 7.37 15.3 R. 15,-5,0 P-O 45" L	37 7.40 16.2 R. 15,0 P-O 65" L	41 7.35 15.7 R. 15,-5,0 P-O 85" L
M13*	22 7.56 16.9 Before Anaesthesia	21 7.55 17.2 H.S. 90", P-O 35" L	18 7.58 18.0 R. 15,0 P-O 60" L	17 7.39 18.2 R. 15,-5,0 P-O 85" L	15 7.61 18.6 R. 15,0,-5 P-O 110" L
M14*	27 7.45 15.3 H.S. 50" S	21 7.52 16.6 R. 15,0 35" S	14 7.64 19.2 R. 15,-5 P-O 135" L 21 7.50 16.2 R. 15,-5,0 30" S	21 7.48 20.0 H.S. P-O 180" L 21 7.50 16.8 R. 15,0,-5 30" S	17 7.56 20.1 R. 15,-5,0 P-O 210" L 25 7.42 18.7 R. 15,0 P-O 30" L 32 7.35 18.8 R. 15,-5,0 P-O 60" L
M15	32 7.39 14.4 Before Anaesthesia	18 7.58 14.9 R. 15,-5 30" L	18 7.58 14.5 R. 15,-5 P-O 30" L	19 7.54 14.1 R. 15,-5 P-O 60" L	
M16	33 7.42 10.8 Before Anaesthesia	29 7.42 11.4 R. 15,-5 30" L	51 7.10 12.4 R. 15,-5 P-O 30" L	22 7.58 13.4 R. 15,-5 P-O 65" L	29 7.41 12.4 R. 15,-5 30" L

* See Figures

Abbreviations: H.S.: Hand Squeeze R.: Respirator P-O: Pleura Open ": Minutes
S: Supine position L.: Lateral position P.: Prone position

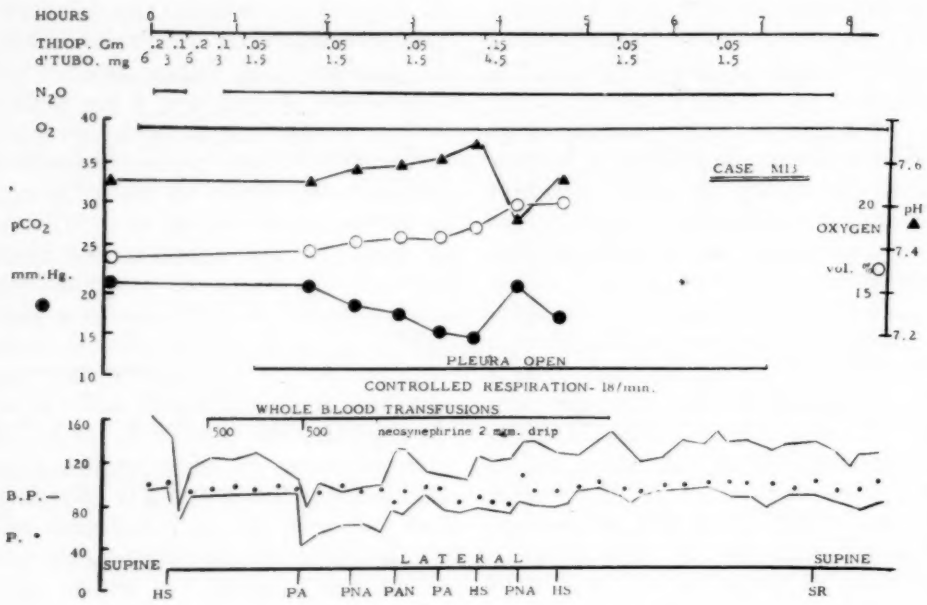

FIG. 3

Chart M13.—Male, white, age 78 with mild hypertensive cardiovascular disease and carcinoma of lower third of oesophagus.

Premedication: Pethidine 50 mg, atropine 0.6 mg.

Operation: Resection of lower third of oesophagus and oesophago-jejunostomy.

Anaesthesia time: 8 hours 5 minutes.
Anaesthetic agents: N_2O (50%); thiopentone 1,000 mg; d-tubocurarine 30 mg.

Note. Before induction of anaesthesia arterial pCO_2 22 ●; pH 7.56 ▲ : O_2 content 16.9 vol. % O.

On induction, blood pressure dropped steeply from 160/86 to 78/? Posturing in head low position assisted in prompt return to stable level of 120/82. Then patient was turned to lateral position. Respiration was controlled by hand squeeze (HS) for 90 minutes. During the last 30 minutes pleura was open and systolic pressure slid down slowly. Then the respirator was turned on with pressures of +15 mm Hg to 0, 1:2 (PA). Blood pressure promptly dropped again to 78/40 and then stabilized at a slightly higher level. Twenty minutes later pattern was changed to +15 mm Hg, −5 mm Hg, to 0 (1:1:1) (PNA). This did not help in restoring the blood pressure after 10 minutes, and phenylephrine HCl was added to the infusion. Ten minutes later the pattern was changed to +15 mm Hg; 0 (atmospheric pressure); and −5 mm Hg (NPA). This caused a prompt elevation in both the systolic and diastolic pressure. Changing to +15 mm Hg and atmospheric (PA) again lowered the blood pressure. Subsequently HS, PNA and HS were used with only moderate changes of the blood pressure. During the period the pleura was open seven arterial blood samples were drawn, each at the end of a 20-minute period of HS or respirator pattern as noted. Throughout this time the pCO_2 fell gradually, pH rose slowly except at the end of the second period of HS, and the oxygen content rose slowly.

These indicated that effective alveolar ventilation was being provided during the intrathoracic operation, while cardiovascular homeostasis was disturbed by positive pressure breathing.

Fluids during surgery: 1000 ml whole blood, 1000 ml 5% D/W, 500 ml N/S.

Supportive therapy: 2 mg phenylephrine HCl added to 250 mg whole blood administered from 165 min to 315 min by very slow drip. Postanaesthetic course was uneventful.

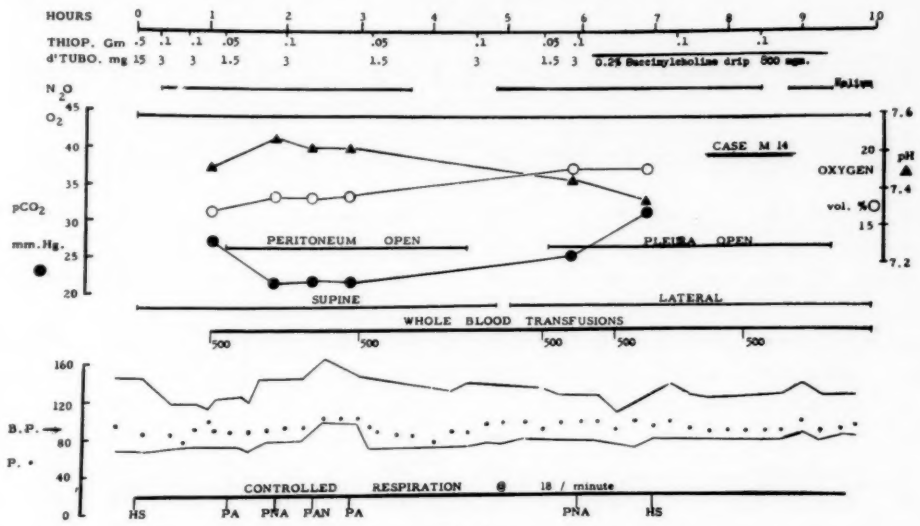

FIG. 4

Chart M 14.—Male, black, age 54, height 178 cm, weight 80 kg, with syphilitic aortic insufficiency and carcinoma of the middle third of the oesophagus.

Premedication: Meperidine 75 mg; atropine 0.4 mg.

Operation: Abdominal exploration and freeing of lower end of oesophagus; thoracotomy and resection of middle third of oesophagus.

Anaesthesia time: 9 hours 30 minutes.

Anaesthetics: N_2O (50%), thiopentone 1350 mg, d-tubocurarine 34.5 mg, suxamethonium 800 mg.

Note. This operation was done in two stages. Before the abdominal cavity was opened, arterial pCO_2 27 ●; pH 7.45 ▲; O_2 content 15.3 vol. % ○.

Blood pressure descended slowly with induction of anaesthesia and with PPB by hand squeeze (HS). When respirator was used, PA caused no significant change, while addition of a subatmospheric phase elevated the blood pressure (PNA and NPA). This was lowered by switching back to PA, but remained quite stable thereafter. When the patient was turned lateral and the pleura was opened (at 340 minutes) +15 mm Hg, −5 mm Hg, 0 (atmospheric pressure) (1:1:1) at 18/min was used for 30 minutes followed by HS for the remainder of the operation without significant change in the blood pressure. During the period the peritoneum and pleura were open 5 arterial blood samples were drawn. pCO_2, pH and O_2 content remained within normal limits throughout.

Fluids during surgery: 2,500 ml whole blood; 1,700 ml 5% D/W, 700 ml N/S.

Supportive therapy: Diphenhydramine 25 mg iv (75 min) cough on tube; atropine 0.4 mg (135 min); suxamethonium 20 mg (230 min)—hiccough; atropine 0.4 mg (375 min); atropine 0.2 mg (450 min); helium-oxygen (570–660 min); oxygen 100% (660–750 min); nasal oxygen for 2 hours postoperatively.

Postanaesthetic course was uneventful.

the 3-phase system of +15 mm Hg/−5 mm Hg/atmospheric pressure with phases of equal duration. This pattern was troublesome in 6 of the 8 patients (tables VII (b) and VIII (b)). The other disturbing pattern was that employing the 2-phase system of +15 mm Hg/−5 mm Hg with phases of equal duration. In 7 of the 8 patients this pattern was troublesome (tables VII (d) and VIII (d)). This was unfortunate in the latter group, as it appeared both from the blood data (the pCO_2 and the pH) and clinically that the patient could be maintained most easily

TABLE III
Pneumonectomy operations.

Pt	Age	Sex	Color	S A	Physical State	Premed		Position	Anaesthetics Thiop	dTc	N_2O	Controlled Respiration Method	Pattern mm Hg	Phase Time Ratio	Blood Trans.	Anaes Time	Pleura Open
P1	68	M	W	1.72	3	M75	A.6	L	450 Mep. 150	21	75%	HS	+10-15,0	1:2	1500	260	165
P2	69	M	W	1.76	3	M75	A.6	L	600		50%	Ohio	+15,0	1:2	1000	295	210
P3	38	M	B	1.93	3	C50	A.6	L	500	21	50%	HS	+10-15,0	1:2	2000	270	180
P4	49	M	W	1.76	3	C50	A.6	L	1050	27	50%	HS	+10-15.0	1:2	1500	360	245
P5	23	M	B	1.84	2	C50 M55	S.6	L	675	21	50%	HS	+10-15,0	1:2	1500	300	195
P6	51	@* F	W	1.45	4	C25	S.6	L	875	24	50%	HS	+10-15,0	1:2	4000	360	240
P7	21	M	W	1.79	2	C25	S.6	L	1500	45	50%	HS	+10-15.0	1:2	4500	310	220
P8	38	F	B	1.65	4	C25	S.6	L	750	23	50%	Blease	+15,0	1:2	1500	240	120
P9	54	M	W	1.67	3	C25	S.6	L	700	21	50%	Blease	+15,0	1:2	2500	320	210
P10	46	M	W	1.85	3	C25	S.6	L	650	27	50%	HS	+10-15,0	1:2	2500	300	180
P11	51	M	B	1.62	3	C25	S.6	L	600	23	50%	HS	+10-15,0	1:2	1500	360	250
P12	20	#* F	W	1.42	3	C25	S.6	L	475	17	50%	HS	+10-15,0	1:2	1500	300	195
P13	35	F	W	1.73	3	C25	S.6	L	800	39	50%	HS	+10-15.0	1:2	5000	330	205
P14	44	* M	W	1.75	3	C25	S.6	L	500	30	50%	HS	+10-15,0	1:2	2000	360	250
P15	35	* M	W	1.74	3	C25	S.6	L	800	30	50%	Ohio	+15,0	1:2	2500	450	300
P16	54	M	B	1.56	3	C25	S.6	L	825	25	50%	HS	+10-15,0	1:2	4000	270	170
P17	26	F	W	1.46	3	C25	S.6	L	650	24	50%	HS	+10-15.0	1:2	2000	300	175
P18	34	M	B	1.72	3	C25	S.6	P	600	18	50%	Ohio	+15,-5,0	1:1:1	1000	270	160
P19	34	M	W	1.92	3	C25	S.6	L	650	24	50%	Ohio	+15,0,-5	1:1:1	2000	300	190
P20	18	F	B	1.48	3	C25	S.6	L	400	15	50%	Ohio	+15,0,-5	1:1:1	1500	225	100
P21	19	M	B	2.10	3	C25	S.6	L	900	27	50%	Ohio	+15,0,-5	1:1:1	1500	285	155
P22	34	@ M	B	1.86	3	C25	S.6	L	625	30	50%	Jeff.	+15,-5	1:1	4000	360	245

SA=Surface area sq. metres.

@ Pleural Pneumonectomy
# Decortication
* Thoracoplasty

C=Chlorpromazine.
M=Meperidine.
A=Atropine.
S=Scopolamine.

L=Lateral.
P=Prone.
HS=Hand Squeeze.
Jeff.=Jefferson Respirator

in a near-normal state with these pressures and this phase timing. The only patient among these with a considerable fall in pH (7.19) had severe "trapping" (S25).

Of the 46 patients managed without the negative phase (21 patients with hand squeeze and 25 patients with +15 mm Hg/atmospheric pressure) lung inflation was satisfactory in 19 and the surgeon was not hindered by the movements in the field. In the other 27 cases the anaesthetist noted, or the surgeon often complained of, either inadequate inflation of the lung or hindering movements in the field. Five among these, managed with the respirator together with their blood data, are noted in tables VII (a) and VIII (a). In three of these five patients moderate falls in pH

Lobectomy operations.

Pt.	Age	Sex	Colour	SA	Physical state	Premedication	Position	Anaesthetics			Controlled respiration			Blood trans.	Anaes. time min	Pleura open min
								Thiop.	dtc	N_2O	Method	Pattern mm Hg	Phase : time ratio			
L1	13	M	B	1.20	2	M15	A.6	L	500	12	50%	HS Ohio	15, 0 15, −5, 0	1:2 1:1:1	500	315	150
L2	51	*M	B	1.73	3	C25 M25	A.6	L	950	27	50%	HS	10−15, 0	1:2	2500	300	185
L3	34	†M	W	1.64	3	C25 M25	A.6	L	825	24	50%	HS	10−15, 0	1:2	1500	300	170
L4	34	M	B	1.86	3	C50 M25	A.6	L	700	21	50%	HS	10−15, 0	1:2	1000	240	120
L5	31	M	W	1.66	3	C50 M25	A.6	L	1500	45	50%	Blease	15, 0	1:2	2000	330	195
L6	36	M	W	1.80	3	C50 M25	A.6	L	1000	30	50%	Blease	15, 0	1:2	1500	240	165
L7	43	†M	B	1.79	3	C50 M25	A.6	L	1000	31	50%	Blease	15, 0	1:2	1500	330	195
L8	47	†M	B	1.57	3	C25 M25	S.6	L	850	26	50%	Blease	15, 0	1:2	1000	255	180
L9	12	F	B	1.52	2	C25	A.4	L	400	15	50%	HS	10−15, 0	1:2	1000	300	190
L10	31	F	B	1.80	3	C50	A.6	L	400	30	50%	HS	10−15, 0	1:2	1000	300	185
L11	43	†M	B	1.92	3	C25	A.6	L	1000	30	50%	HS	10−15, 0	1:2	2000	420	295
L12	30	†F	B	1.63	3	C50	A.6	L	675	18	50%	HS	10−15, 0	1:2	2000	330	205
L13	48	†F	B	1.76	3	C25	A.6	L	1175	30	50%	HS	10−15, 0	1:2	1500	390	240
L14	42	F	W	1.59	3	C50	A.6	L	925	24	50%	HS	10−15, 0	1:2	3500	330	200
L15	53	M	B	1.94	3	C50	A.6	L	1000	33	50%	HS	10−15, 0	1:2	2500	435	315
L16	18	F	W	1.61	3	C25	S.6	L	650	19	50%	Blease	15, 0	1:2	1000	240	150
L17	36	‡F	W	1.60	3	C25	S.6	L	700	21	50%	Blease	15, 0	1:2	2500	360	240
L18	53	†M	W	1.50	3	C25	S.6	L	550	17	50%	Blease	15, 0	1:2	1000	255	165
L19	22	M	W	1.76	3	C25	S.6	L	780	21	50%	Blease	15, 0	1:2	1500	360	180
L20	39	F	W	1.65	3	C25	S.6	L	550	19	50%	HS	10−15, 0	1:2	500	210	135
L21	34	F	B	1.57	3	C25	S.6	L	700	26	50%	Blease	15, 0	1:2	1500	285	150
L22	19	†M	B	1.94	3	C25	S.6	L	800	30	50%	HS	10−15, 0	1:2	1000	270	155
L23	42	M	W	1.81	3	C25	S.6	L	850	25	50%	HS	10−15, 0	1:2	1500	240	140
L24	40	M	W	1.90	3	C25	S.6	L	725	27	50%	HS	10−15, 0	1:2	2000	360	250
L25	41	M	B	1.88	3	C25	S.6	L	860	36	50%	HS	10−15, 0	1:2	2500	330	205
L26	38	*M	B	1.88	3	C25	S.6	L	800	30	50%	HS	10−15, 0	1:2	2000	420	300
L27	61	†M	B	1.75	3	C25	S.6	L	600	25	50%	HS	10−15, 0	1:2	2000	330	195
L28	49	†M	B	1.95	3	C25	S.6	L	800	30	50%	HS	10−15, 0	1:2	3000	390	260
L29	24	*†M	W	1.72	3	C25	S.6	L	650	25	50%	HS	10−15, 0	1:2	2000	420	305
L30	41	‡†M	B	1.51	3	C25	S.6	L	575	19	50%	HS	10−15, 0	1:2	1000	330	200
L31	63	*†M	W	1.92	4	C25	S.6	L	900	42	50%	Ohio	15, 0	1:2	3500	450	300
L32	53	M	W	2.01	3	C25	S.6	L	750	24	50%	Ohio	15, 0	1:2	2000	330	195
L33	30	F	W	1.52	3	C25	S.6	L	750	27	50%	Ohio	15, −5, 0	1:1:1	1000	270	140
L34	54	M	B	1.92	3	C25	S.6	L	750	21	50%	Ohio	15, −5, 0	1:1:1	1500	450	240
L35	38	M	B	2.07	3	C25	S.6	L	1000	30	50%	Ohio	15, −5, 0	1:1:1	2000	330	160
L36	53	M	W	1.94	3	C25	S.6	L	800	30	50%	Ohio	15, −5, 0	1:1:1	1500	330	180
L37	34	M	B	1.88	3	C25	S.6	L	725	27	50%	Ohio	15, −5, 0	1:1:1	1500	270	145
L38	61	M	W	1.58	3	C25	S.6	P	900	27	50%	Ohio	15, −5, 0	1:1:1	2500	360	205
L39	37	M	W	2.02	3	C25	S.6	L	900	27	50%	Ohio	15, 0, −5	1:1:1	1000	255	130
L40	21	F	B	1.65	3	C25	S.6	L	850	25	50%	Ohio	15, −5	1:1	2000	315	210
L41	36	M	W	1.88	3	C25	S.6	L	800	27	50%	HS	10−15, 0	1:2	1500	285	185
L42	26	M	B	1.61	3	C25	S.6	L	600	27	50%	Ohio	15, −5	1:1	1000	270	140
L43	24	F	W	1.52	2	C25	S.6	L	800	33	50%	Jeff.	15, −5	1:1	1000	240	160
L44	23	F	W	1.42	3	C25	S.6	L	600	27	50%	Jeff.	15, −5	1:1	750	280	145

S.A. = Surface area sq. metres.
* Thoracoplasty.
‡ Decortication
† Partial pneumonectomy (lobe and segments).
C = Chlorpromazine.
M = Meperidine.
A = Atropine.
S = Scopolamine.
L = Lateral.
P = Prone.
HS = Hand squeeze.
Jeff. = Jefferson Respirator.

TABLE V
Segmental resection operations.

Pt.	Age	Sex	Color	S.A.	Physical State	Premedication	Position	Anaesthetics Thiop.	dTc	N_2O	Controlled Respiration Method	Pattern mm Hg	Phase Time Ratio	Blood Trans.	Anaes. Time Min.	Pleura Open Min.
S1	32	F	W	1.77	3	C25 M25 A.6	L	850	27	50%	HS	10-15,0	1:2	1000	225	140
S2	19	F	W	1.30	2	C50 A.6	L	500	15	50%	HS	10-15,0	1:2	2000	195	105
S3	35	F	W	1.92	2	C50 A.6	L	1000	42	50%	HS	10-15,0	1:2	1000	330	220
S4	28	M	W	1.98	2	C50 A.6	L	850	24	50%	HS	10-15,0	1:2	1000	255	120
S5	60	M	W	1.80	3	C50 A.6	L	750	24	50%	HS	10-15,0	1:2	1000	270	145
S6	16	M	W	1.75	2	C25 S.6	L	360	6	50%	HS	10-15,0	1:2	500	210	110
S7	63	M	W	1.73	3	C25 S.6	L	650	24	50%	HS	10-15,0	1:2	500	210	120
S8	44	M	B	1.72	3	C25 S.6	L	400	15	50%	HS	10-15.0	1:2	0	135	50
S9	15	F	W	1.46	2	C25 S.6	L	375	700 Sc	50%	HS	10-15,0	1:2	500	180	90
S10	32	M	W	1.94	2	C50 M25 S.6	L	500	24	50%	Blease	15,0	1:2	500	195	90
S11	37	F	W	1.59	2	C50 M25 S.6	L	700	27	50%	Blease	15.0	1:2	1500	270	165
S12	42	M	B	1.91	3	C25 S.6	L	900	30	50%	Blease	15,0	1:2	1500	320	195
S13	26	M	B	1.63	3	C25 S.6	L	800	30	50%	Blease	15,0	1:2	2000	330	255
S14	53	M	B •	1.96	2	C25 S.6	L	1100	30	50%	Blease	15.0	1:2	1500	360	255
S15	44	M	B	1.91	2	C25 S.6	L	1050	37	50%	Blease	15,0	1:2	2500	360	240
S16	48	M	W	1.93	3	C25 S.6	L	700	26	50%	Blease	15,0	1:2	1000	255	165
S17	25	M	W	1.87	3	C25 S.6	L	825	25	50%	Blease	15,0	1:2	1500	270	180
S18	26	F	B	1.60	3	C25 S.6	L	800	30	50%	Blease	15,0	1:2	1000	300	195
S19	35	M	W	1.68	3	C25 S.6	L	650	24	50%	Ohio	15,0	1:2	1000	330	215
S20	49	M	W	1.81	3	C25 S.6	L	1000	30	50%	Ohio	15,0	1:2	2000	330	210
S21	30	M	B	1.48	3	C25 S.6	L	480	18	50%	Ohio	15,-5,0	1:1:1	1500	315	190
S22	41	M	W	1.98	3	C25 S.6	L	1100	47	50%	Ohio	15,0,-5	1:1:1	2000	420	260
S23	28	M	W	1.83	3	C25 S.6	L	725	43	50%	Ohio	15,0,-5	1:1:1	1000	335	195
S24	40	M	W	1.72	3	C25 S.6	L	560	17	50%	Ohio	15,0,-5	1:1:1	1000	230	110
S25	31	F	W	1.69	2	C25 S.6	L	800	24	50%	Ohio	15,-5	1:1	1000	255	140
S26	54	M	B	1.94	3	C25 S.6	L	700	21	50%	Ohio	15,-5	1:1	1000	270	145
S27	36	F	W	1.44	2	C25 S.6	P	375	24	50%	Jeff.	15,-5	1:1	1000	250	130

SA=Surface area sq. metres. • Thoracoplasty C=Chlorpromazine. A=Atropine.
Jeff.=Jefferson Respirator. @ Multiple Segments M=Meperidine. S=Scopolamine.

were observed and could not be adequately corrected during the operation.

For operating conditions and considering the acid-base homeostasis, it was felt that the least satisfactory pattern in this study was that employing the 3-phase system of + 15 mm Hg/ - 5 mm Hg/atmospheric pressure with each phase of equal duration. All but one patient had fair pulmonary function and were comparable to those in the other groups. The remaining patient (L35) had a considerable disturbance in pH (7.35 to 7.08) and pCO_2 (38 to 84) probably due to severe "trapping" (expiration time was 7 seconds).

Clinically and for the surgeon the most

TABLE VI

Pulmonary function studies—patients for controlled respiration with Blease pulmoflator.

Patient	Surface Area	Maximum Breathing Capacity Litres/min.	Walking Ventilation M.B.C.	Total Vital Capacity Litres	Timed Vital Capacity 1" litres	2" litres	3" litres	Expiration Time Seconds	Bronchospirometry Minute Volume %	Oxygen Consumption %	Vital Capacity %	Radford Predicted Tidal Volume Litres
P8	1.65	58(53)*	.34	1.50 (50)	1.14 (76)	1.31 (88)	1.43 (96)	4.0				.350
P9	1.67	89(71)	19	2.08 (50)	1.64 (79)	1.85 (89)	1.98 (97)	5.0	R. 20 L. 80	14 86	32 68	.350
L5	1.66	85(54)	19	2.82 (64)	2.04 (73)	2.34 (83)	2.46 (88)	7.0	R. 45 L. 55	45 55	45 55	.350
L6	1.80	125(81)	13	4.25 (97)	3.18 (77)	3.56 (82)	3.61 (85)	6.0	R. 46 L. 54	50 50	49 51	.400
L7	1.79	123(85)	17	4.15 (93)	3.50 (84)	3.62 (87)	3.72 (90)	4.5	R. 44 L. 56	41 59	40 60	400
L8	1.57	77(62)	.22	2.10 (56)	1.57 (75)	1.70 (81)	1.74 (83)	5.0	R. 46 L. 54	44 56	44 56	.350
L16	1.61	128(102)	.12	3.85 (124)	2.78 (75)	3.53 (92)	3.72 (97)	4.0				.350
L17	1.60	104(90)	.10	3.20 (102)	2.70 (84)	2.90 (96)	3.20 (100)	3.1	R. 40 L. 60	36 64	32 68	.350
L18	1.51	68(56)	.24	1.86 (79)	1.19 (64)	1.25 (67)	1.32 (71)	9.0	R. 75 L. 25	83 17	75 25	325
L19	1 76	138(81)	.13	3.90 (89)	2.48 (64)	3.28 (85)	3.47 (89)	5.0				375
L21	1 57	104(83)	.19	3.99 (133)	3.52 (76)	3.34 (84)	3.48 (87)	5.0				350
S10	1 94			4.18 (97)				2.5				.550
S11	1 59	112(90)	.15	2.98 (99)	2.60 (87)	2.77 (93)	2.86 (96)	7.5	R. 53 L. 47	53 47	52 48	375
S12	1.91	78(50)	23	3.18 (61)	2.02 (64)	2.34 (74)	2.47 (78)	9.0	R. 50 L. 50	55 45	54 46	425
S13	1.63	85(60)	20	2.09 (52)				4.0				350
S14	1.96	114(82)	19	3.65 (72)	2.67 (73)	3.02 (79)	3.20 (88)	7.0	R. 66 L. 34	70 30	69 31	.450
S15	1.91	121(79)	12	4.66 (96)	3.16 (68)	3.55 (76)	3.62 (80)	5.0	R. 52 L. 48	50 50	51 49	.400
S16	1.93			4.05 (103)				3.0				.450
S17	1 87	137(78)	.13	4.96 (106)	2.24 (45)	4.00 (80)	4.35 (88)	8.0				.425
S18	1 60	112(93)	.12	3.18 (107)	2.61 (82)	3.10 (98)	3.18 (100)	5.0				.350

* Figures in brackets represent percent of predicted normal

satisfactory pattern employed was the 3-phase system of +15 mm Hg/atmospheric pressure/−5 mm Hg, with each phase of equal duration (tables VII (c) and VIII (c)). During these operations there was no complaint by the surgeon of hindering movements in the operative field, and in only one of the 7 cases (P20) was there significant fall in the pH while the pleura was open. This corrected itself as the operation progressed.

Although it may be invalid to draw

TABLE VII(*a*)

Patient	Surface Area	Maximum Breathing Capacity Litres/min.	Walking Ventilation M.B.C.	Total Vital Capacity Litres	Timed Vital Capacity 1" litres	2" litres	3" litres	Expiration Time Seconds	Bronchospirometry Minute Volume %	Oxygen Consumption %	Vital Capacity %	Radford Predicted Tidal Volume Litres
P15	1.74	72(44)*	.20	2.81 (64)	1.96 (70)	2.33 (83)	2.48 (88)	5.0	R. 90 L. 10			.375
L31	1.92	85(79)	.29	3.61 (78)	2.71 (75)	3.31 (92)	3.61 (100)	3.0	R. 74 L. 26	80 20	73 27	.450
L32	2.01	125(90)	.16	5.24 (105)	3.80 (73)	4.45 (85)	4.80 (92)	5.5	R. 40 L. 60			.450
S19	1.68	140(88)	.10	3.84 (93)	2.24 (58)	3.18 (83)	3.53 (92)	5.0	R. 62 L. 38			.350
S20	1.81	100(75)	.18	3.05 (70)	1.61 (53)	2.15 (70)	2.43 (80)	5.0	R. 45 L. 55			.400

*Figures in brackets represent percent of predicted normal

TABLE VII(*b*)

Patient	Surface Area	Maximum Breathing Capacity Litres/min.	Walking Ventilation M.B.C.	Total Vital Capacity Litres	Timed Vital Capacity 1" litres	2" litres	3" litres	Expiration Time Seconds	Bronchospirometry Minute Volume %	Oxygen Consumption %	Vital Capacity %	Radford Predicted Tidal Volume Litres
P18	1.72	109(66)*	.16	3.06 (72)	2.18 (72)	2.70 (88)	2.88 (94)	5.5	R. 79 L. 21	82 18	80 20	.350
L33	1.52	76(71)	.20	1.93 (70)	1.35 (69)	1.59 (81)	1.95 (100)	3.0	R. 48 L. 52	41 59	45 55	.325
L34	1.92	120(78)	.19	4.00 (88)	2.98 (69)	3.40 (87)	3.75 (93)	5.0	R. 70 L. 30			.450
L35	2.07	148(90)	.13	4.95 (95)	4.05 (82)	4.46 (90)	4.68 (95)	7.0	R. 40 L. 60			.500
L36	1.94	122(93)	.27	4.10 (95)	3.51 (86)	3.92 (96)	3.96 (97)	4.5	R. 40 L. 60			.500
L37	1.88	127(79)	.14	3.58 (77)	3.27 (90)	3.48 (97)	3.56 (99)	3.5	R. 35 L. 65			.450
L38	1.58	110(99)	.16	3.60 (86)	2.37 (66)	2.85 (79)	3.11 (86)	5.0	R. 38 L. 62			.350
S21	1.48	118(78)	.14	2.80 (77)	2.40 (76)	2.62 (94)	2.64 (95)	3.0	R. 68 L. 32			.325

*Figures in brackets represent percent of predicted normal

Pulmonary function studies—patients managed entirely with experimental respirator.

TABLE VII(*c*)

Patient	Surface Area	Maximum Breathing Capacity Litres/min.	Walking Ventilation M.B.C.	Total Vital Capacity Litres	Timed Vital Capacity 1" litres	2" litres	3" litres	Expiration Time Seconds	Bronchospirometry Minute Volume %	Oxygen Consumption %	Vital Capacity %	Radford Predicted Tidal Volume Litres
P19	1.92	90(53)*	.20	2.48 (51)	1.85 (75)	2.06 (83)	2.26 (91)	7.0	R. 84 L. 16	90 10	85 15	.450
P20	1.48	75(62)	.23	1.58 (56)	1.34 (85)	1.44 (91)	1.49 (94)	5.0	R. 90 L. 10	95 5	91 9	.325
P21	2.10	147(70)	.13	4.10 (78)	2.24 (57)	3.18 (78)	3.48 (85)	5.0	R. 68 L. 32	82 18	69 31	.500
L39	2.02	122(71)	.16	4.05 (82)	2.75 (68)	3.28 (79)	3.54 (88)	4.5	R. 40 L. 60			.500
S22	1.98	127(85)	.15	4.80 (100)	3.80 (79)	4.24 (88)	4.51 (94)	3.5	R. 49 L. 51	40 60	48 52	.500
S23	1.83	71(42)	.21	2.92 (61)	2.10 (72)	2.36 (81)	2.51 (86)	7.0	R. 65 L. 35	81 19	65 35	.425
S24	1.72	141(94)	.12	4.08 (97)	3.01 (74)	3.44 (84)	3.73 (91)	4.0	R. 62 L. 38			.375

*Figures in brackets represent percent of predicted normal

TABLE VII(*d*)

Patient	Surface Area	Maximum Breathing Capacity Litres/min.	Walking Ventilation M.B.C.	Total Vital Capacity Litres	Timed Vital Capacity 1" litres	2" litres	3" litres	Expiration Time Seconds	Bronchospirometry Minute Volume %	Oxygen Consumption %	Vital Capacity %	Radford Predicted Tidal Volume Litres
P22	1.86	100(72)*		2.94 (67)	1.95 (66)			4.5	R. 72 L. 28	100 0	88 12	.450
L40	1.65	109(91)	.13	2.16 (72)	1.64 (71)	1.86 (86)	1.94 (90)	4.0	R. 45 L. 55			.375
L42	1.61	105(82)	.17	2.64 (65)	1.82 (70)	2.41 (91)	2.52 (95)	5.0	R. 45 L. 55			.350
L43	1.52			2.83 (93)				3.0				.325
L44	1.42	34(38)		2.96 (100)	0.81 (27)			5.6				.300
S25	1.69	96(83)	.17	2.47 (79)	1.51 (61)	1.82 (74)	1.94 (79)	10.0	R. 72 L. 28	76 24	72 28	.375
S26	1.94	102(78)	.21	3.74 (77)	2.80 (75)	3.38 (92)	3.50 (93)	3.0	R. 58 L. 42	58 42	62 38	.475
S27	1.44			2.75 (98)				2.5				.300

* Figures in brackets represent percent of predicted normal

Pulmonary function studies—patients managed entirely with experimental respirator.

TABLE VIII

Arterial blood data from patients managed entirely with experimental respirator.

	Respirator Pattern	Patient	Before Anaesthesia pCO_2 mm.Hg.	pH	O_2 vol.%	Steady Anaesthesia Positioned 30 min. pCO_2 mm.Hg.	pH	O_2 vol.%	Pleura Open 30 min. pCO_2 mm.Hg.	pH	O_2 vol.%	Pleura Open 90 min. pCO_2 mm.Hg.	pH	O_2 vol.%	Chest Closed 30 minutes pCO_2 mm.Hg	pH	O_2 vol.%
(a)	15, 0 1:2 @ 18/min.	P15*	31	7.45	16.1	47	7.33	16.5	51	7.29	17.9	37	7.40	16.6	37	7.37	15.5
									Pleura open 180 min.→			35	7.41	17.2			
		L31	24	7.49	19.6	26	7.46	21.8	43	7.27	19.6	38	7.30	18.4	45	7.27	17.7
		L32	-		18.9	22	7.55	19.2	24	7.51	17.9	25	7.48	17.9	23	7.42	18.9
		S19	40	7.35	19.7	28	7.43	19.0	26	7.43	19.3	23	7.44	20.6	44	7.24	21.8
									Pleura open 180 min.→			31	7.35	21.9			
		S20	38	7.35	17.3	31	7.42	20.1	55	7.21	20.1	55	7.21	19.3	47	7.25	17.7
		AVERAGE	32	7.41	18.7	30	7.44	19.3	40	7.34	19.0	36	7.37	18.6	40	7.31	18.3
(b)	15, -5, 0 1 : 1 : 1 @ 18/min	P18*	36	7.35	16.3	33	7.38	18.7	36	7.32	17.3	35	7.30	17.4	43	7.22	17.4
		L33	38	7.38	17.1	29	7.45	17.2	34	7.39	18.3	48	7.28	17.8	40	7.34	16.3
		L34	36	7.38	18.4	25	7.51	18.2	26	7.47	17.9	35	7.36	17.5	37	7.33	18.6
		L35	37	7.35	18.1	38	7.39	19.6	41	7.30	18.5	84	7.08	18.4	75	7.11	18.2
		L36	29	7.44	18.7	27	7.45	21.3	37	7.31	20.8	34	7.38	21.3	35	7.33	19.9
		L37	33	7.32	20.0	36	7.40	21.0	-			35	7.29	20.0	41	7.22	18.4
		L38	28	7.44	17.9	27	7.41	19.4	20	7.30	18.0	23	7.37	17.0	31	7.35	18.0
		S21	33	7.40	20.0	18	7.58	21.3	21	7.49	21.5	23	7.39	21.5	41	7.19	19.6
		AVERAGE	34	7.38	18.3	28	7.45	19.6	32	7.37	18.9	40	7.31	18.9	42	7.26	18.3
(c)	15, 0, -5 1 : 1 : 1 @ 18/min.	P19*	30	7.38	19.2	31	7.36	18.9	34	7.33	17.0	38	7.31	17.4	38	7.30	17.2
		P20	32	7.41	12.2	35	7.39	12.8	58	7.18	12.4	38	7.34	13.6	37	7.35	13.9
		P21	40	7.31	16.5	32	7.40	19.6	36	7.35	16.6	41	7.31	17.0	45	7.26	18.2
		L39	36	7.39	18.5	31	7.41	20.8	39	7.34	17.9	39	7.32	17.4	36	7.32	17.7
		S22	23	7.54	19.0	22	7.51	20.8	21	7.54	19.2	34	7.36	18.5	35	7.35	12.8
		S23	35	7.38	21.7	30	7.42	21.5	31	7.38	20.9	32	7.36	21.3	31	7.36	20.7
		S24	40	7.36	17.9	27	7.48	17.5	-			28	7.47	17.0	-		17.2
		AVERAGE	34	7.39	17.9	31	7.42	18.8	36	7.35	17.3	36	7.35	17.5	37	7.32	16.8
(d)	15, -5 1:1 @ 18/min.	P22*	38	7.36	17.7	39	7.36	17.4	38	7.38	19.1	47	7.31	17.5	33	7.45	14.9
									Pleura open 180 min.→			46	7.31	13.7			
		L40	35	7.37	15.4	18	7.57	16.7	20	7.53	16.7	47	7.24	16.4	31	7.37	16.9
		L42	27	7.45	17.0	19	7.54	17.4	43	7.24	15.3	29	7.39	17.3	25	7.42	17.4
		L41	-	-	-	21	7.57	19.5	-			18	7.62	14.3	23	7.48	13.5
		L44	38	7.41	14.1	27	7.60	19.2	26	7.62	19.4	26	7.57	19.7	32	7.40	17.1
		S25	27	7.49	17.5	38	7.37	17.7	61	7.19	17.1	35	7.36	17.4	40	7.35	17.6
		S26	27	7.45	17.3	17	7.65	18.7	17	7.51	18.4	21	7.53	16.6	32	7.38	17.7
		S27	36	7.34	16.2	20	7.50	17.6	-			21	7.46	17.1	19	7.54	16.9
		AVERAGE	33	7.41	16.5	32	7.52	18.0	38	7.41	17.7	33	7.44	17.0	31	7.42	16.5

* See Figures

conclusions from this limited and preliminary study, the data indicate the necessity for an extensive trial of the 3-phase system, employing one-third of the cycle for each phase, the rest phase of atmospheric pressure separating inflation from deflation. This type of cycling could avoid the theoretical possibility of increased air trapping due to collapsing bronchioles (Waltz et al., 1954) in patients with extensive disease and a prolonged expiration time; it could avoid the sharp fall of the lung during expiration; and it would allow for a short rapid inflation phase which could provide a wide enough pressure change for adequate pul-

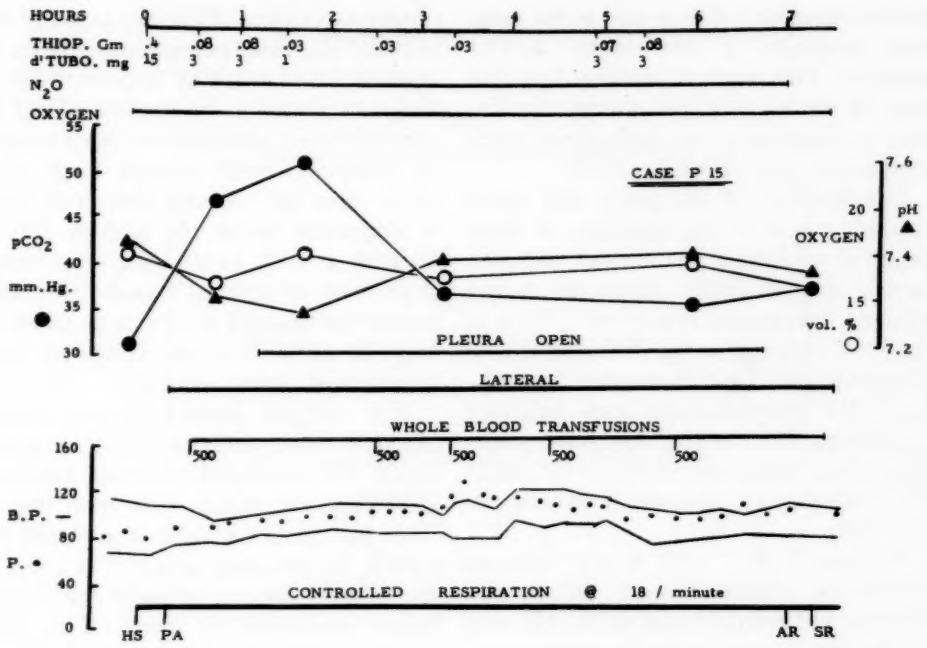

FIG. 5

Chart P15.—Male, white, age 35, height 181 cm; weight 59 kg with non-functioning left lung.

Pulmonary function: MBC 72 l./min (44%); WV 14.3 l./min $\frac{WV}{MBC}$ =0.20 (surgical index).

Vital capacity—Total: 2.81 l.; timed 1″=1.96 l.; 2″=2.33 l.; 3″=2.48 l.

Expiration time: 5 seconds.

Bronchospirometry estimated function: R 90%, L 10%.

Predicted BTV (Radford): 375 ml at 18/min.

Premedication: chlorpromazine 25 mg; scopolamine 0.6 mg.

Operation: Left pneumonectomy and 7 rib thoracoplasty.

Anaesthesia time: 450 minutes; pleura open 310 minutes.

Anaesthetics: N_2O (50%), thiopentone 800 mg, d-tubocurarine 28 mg.

Note. Respiration controlled with mechanical respirator set at +15 mm Hg, 0 (atmospheric pressure) 1:2 at 18/min. Prior to induction of anaesthesia arterial blood pCO_2 31 ●; pH 7.45 ▲; and O_2 content 18.1 vol. % O.

When controlled respiration was instituted, blood pressure was slightly depressed, but remained quite stable throughout the operation. pCO_2 rose early in the operation and then returned to normal levels for the remainder of the operation. The pH followed the pCO_2 changes. The oxygen content dropped early in the operation and remained relatively low, then dropped further after chest closure even though the nitrous oxide was turned off, probably indicative of loss of considerable amount of functioning lung tissue, and at the end the phenomenon of "diffusion anoxia" developing. The surgeon was satisfied with operative conditions throughout the procedure.

Fluids during surgery: 2,500 ml whole blood, 750 ml 5% D/W and 500 ml N/S.

Supportive therapy: Nasal oxygen for 8 hours postoperatively.

Uneventful postanaesthetic course.

monary alveolar inflation and at the same time maintain a low mean airway pressure. This type of cycling has also been shown to assist pulmonary circulation in returning to normal during each respiration cycle (Brecher, 1952).

The stability of the pulse and blood pressure noted in the majority of these patients regardless of airway pressure pattern may be partly attributed to the reduced "vasomotor reactivity" (Price et al., 1954) induced by the chlorpromazine, thiopentone and d-tubocurarine administered for premedication and balanced anaesthesia. Price et al. (1954) have postulated a haemodynamic stimulus by mechanical respirators. This was not evident in our study.

Figures 5, 6, 7 and 8 are redrawn anaesthetic charts showing the cardiovascular and arterial blood pCO_2, pH, and O_2 changes accompanying a pneumonectomy from each group. In each of these patients respiration was controlled by one of the four pressure patterns. Figure 9 represents the average changes in arterial blood pCO_2, pH and O_2 content (volumes per cent) in the four groups of patients managed with the experimental respirator.

There appeared to be a fairly uniform trend in the changes from the pre-anaesthetic blood determinations to the state of stable anaesthesia, the opening of the pleura, and after the chest was closed. In all groups the pCO_2 fell slightly after steady anaesthesia was achieved, and rose after the pleura was opened. The pH rose sharply to a slightly alkalotic level during steady anaesthesia, then fell sharply to a slightly acidotic level when the pleura was opened. In three of the four groups, this level continued downwards even after the pleura was closed. This was possibly due to metabolic acids accumulating from the vigorous movements of hyperventilation, and contributed to by the low pH of the citrated blood administered. Spot samples of relatively fresh citrated bank blood (5-10 days old) reveals that such blood is frequently below pH 6.50 at 37.5°C. In such a study as this one must expect depression of arterial blood pH until a period has elapsed sufficient to allow the body to metabolize the relatively large amounts of citrate injected.

The oxygen content of the arterial blood in these patients rose after anaesthesia was stabilized and then gradually fell while the pleura was open (fig. 9). This fall continued after the pleura was closed. In the third group (+15/0/−5) the oxygen content actually fell slightly below pre-operative levels. This post-operative fall was probably due to the reduction in functioning pulmonary tissue and to the diffusion anoxia phenomenon as noted previously.

Figure 10 graphically shows changes in bicarbonate and pH in the four groups of patients. It should be noted that falls in bicarbonate were usually an accompaniment of elevated pCO_2 (Shaw and Messer, 1932).

It appears evident that this mechanical respirator, which employs fixed pressure cycling, is capable of maintaining a relatively normal pCO_2, even under severe conditions of lung disease. Bjork and Engstrom (1955) noted the same for a mechanical respirator which employs fixed volume cycling. During intrathoracic operations of considerable duration with concurrent administration of citrated whole blood it is probably most difficult

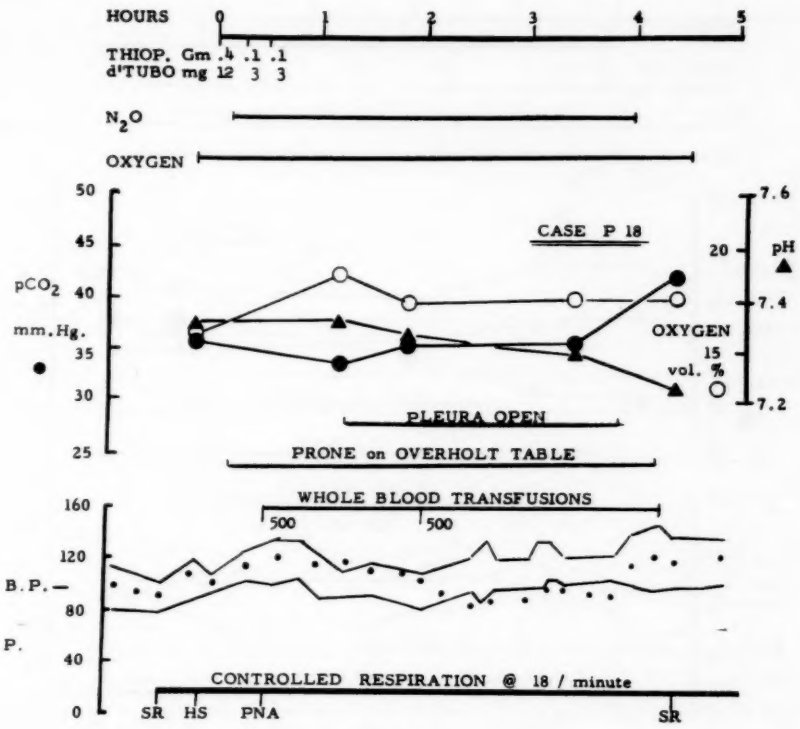

FIG. 6

Chart P18.—Male, black, age 34, height 179 cm, weight 58 kg with nonfunctioning left lung.

Pulmonary function: MBC 109 l./min (66%); WV; 17.4 l./min $\frac{WV.}{MBC}$ =0.16.

Vital capacity: Total 3.06 l.; timed 1″=2.18 l.; 2″=2.70 l.; 3″=2.88 l.

Expiration time: 5.5 seconds.

Bronchospirometry:		MV	O_2C	VC
	R	79	82	86
	L	21	18	14

Predicted BTV (Radford): 350 ml.

Premedication: chlorpromazine 25 mg; scopolamine 0.6 mg.

Operation: Left pneumonectomy.

Anaesthesia time: 270 minutes; pleura open 145 minutes.

Anaesthetics: N_2O (50%); thiopentone 600 mg; d-tubocurarine 18 mg.

Note. Respiration controlled with mechanical respirator set at +15 mm Hg, −5 mm Hg; 0 (atmospheric pressure) 1:1:1: at 18/min. Prior to induction of anaesthesia pCO_2 36 ●; pH 7.35 ▲ and O_2 content 16.3 vol. % O.

Blood pressure remained relatively stable throughout the operation. pCO_2 was adequately cleared throughout, while pH dropped after the pleura was opened, and remained low even after the chest was closed. Oxygen content was satisfactory throughout. The surgeon observed that the respiratory movements were regular throughout, but that the wide swings between inflation and deflation was harassing and interfered with his procedure.

Fluids during surgery: 1000 ml whole blood; 1000 ml 5% D/W; 500 ml N/S.

Supportive therapy: Nasal oxygen 3 hours postoperatively.

Uneventful postanaesthetic course.

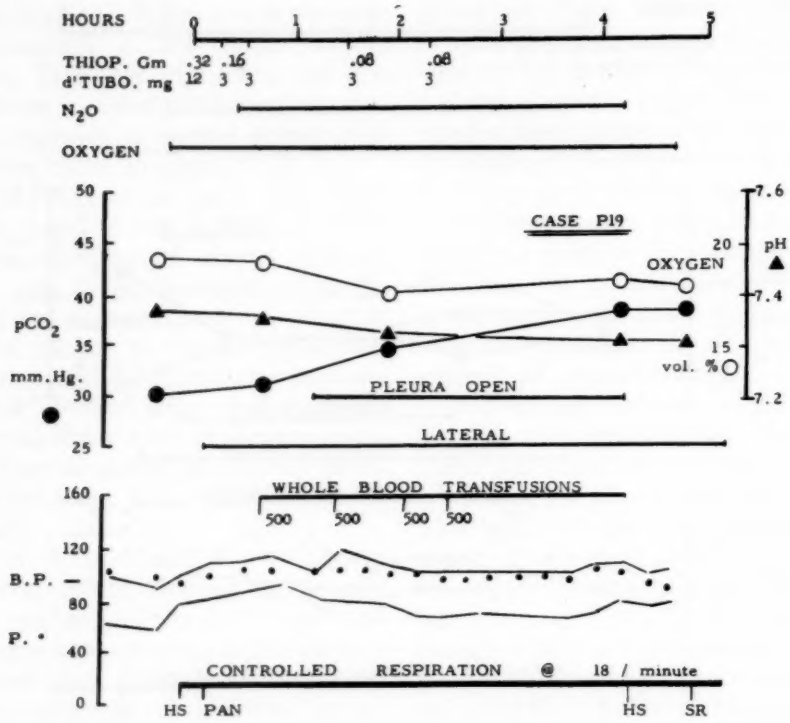

FIG. 7

Chart P19.—Male, white, age 34 height 180 cm, weight 73 kg with nonfunctioning left lung.

Pulmonary function: MBC 90 l./min (53%); WV 19.0 l./min $\frac{WV}{MBC}$ =0.21.

Vital capacity: Total 2.48 l.; timed 1″=1.85 l.; 2″=2.06 l; 3″=2.26 l.

Expiration time: 7 seconds.

Bronchospirometric estimate of function: R 90%, L 10%.

Predicted BTV (Radford): 450 ml at 18/min.

Premedication: chlorpromazine 25 mg; scopolamine 0.6 mg.

Operation: Left pneumonectomy.

Anaesthesia time: 300 minutes; pleura open 190 min.

Anaesthetics: N_2O (50%); thiopentone 650 mg; d-tubocurarine 24 mg.

Note. Respiration controlled with mechanical respirator set at +15 mm Hg, 0 (atmospheric pressure), −5 mm Hg; 1:1:1 at 18/min. Prior to induction of anaesthesia pCO_2 30 ●; pH 7.38 ▲; and O_2 content 19.2 vol. % O.

Blood pressure, pCO_2, pH and O_2 content remained quite stable during the entire operation. The surgeon found this respirator pattern satisfactory throughout the operation.

Fluids during surgery: 2000 ml whole blood, 1000 ml 5% D/W–1000 ml N/S.

Supportive therapy: Nasal oxygen for 6 hours postoperatively.

Uneventful postanaesthetic course.

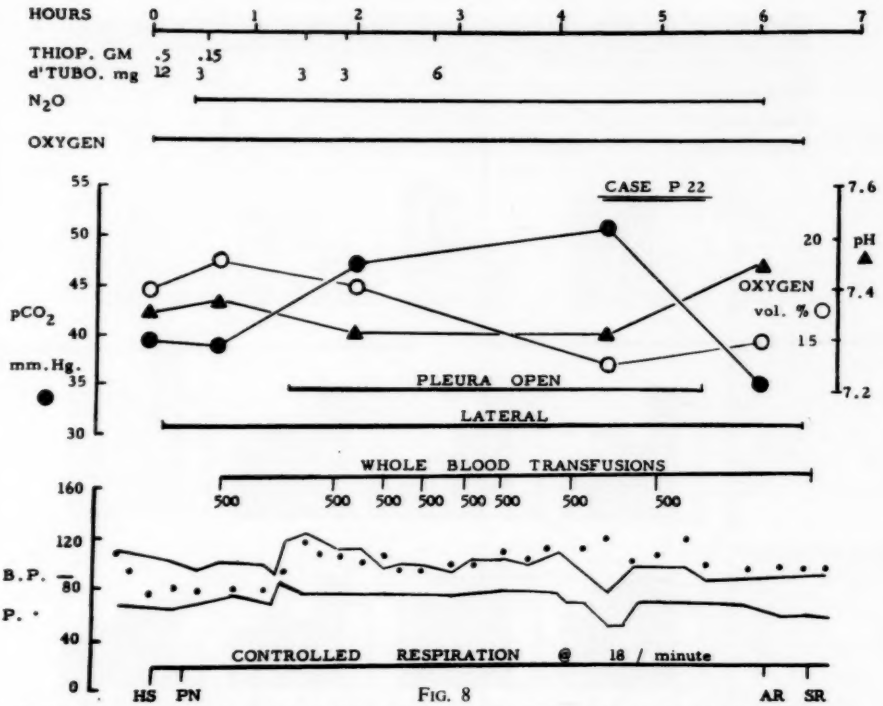

FIG. 8

Chart P22.—Male, black, age 34, height 178 cm, weight 67 kg with non-functioning left lung.
Pulmonary function: Total vital capacity 2.94 l. 1" VC. 1.95 (66%).

Bronchospirometry:							
	R	mv	72%	O_2C	100%	VC	88
	L	mv	28%	O_2C	0	VC	12

Predicted BTV (Radford): 450 ml at 18/min.
Premedication: chlorpromazine 25 mg; scopolamine 0.6 mg.
Operation: left pleural pneumonectomy.
Anaesthesia time: 370 minutes; pleura open 240 minutes.
Anaesthetics: N_2O (50%); thiopentone 625 mg; d-tubocurarine 27 mg.

Note. Respiration controlled with mechanical respirator set at +15 mm Hg; −5 mm Hg 1:1 at 18/min. Prior to induction of anaesthesia pCO_2 38.0 ●; 7.36 ▲ ; O_2 content 17.7 vol. % O.

This patient had very thick pleura which was stuck to chest wall. In the operative procedure continuous marked oozing of blood occurred as the pleura was peeled away from the chest wall. In addition, the anaesthetist was continuously harassed by profuse bronchial secretions which were mucopurulent. After the pleura was opened the airway was frequently disconnected from the respirator to carry out tracheobronchial suctioning. This would account for the moderate elevation of pCO_2 and lowered pH. The blood pressure rose when the pleura was opened, and remained fairly stable except at one point during the fourth hour when blood loss exceeded rate of replacement considerably. When chest was closed pCO_2 and pH were promptly restored to normal levels. The oxygen content also fell to a low level during the difficulty with the pleural dissection, and profuse bronchial secretions and then began to rise when the chest was closed. Here again the surgeon remarked that respiration was quite regular, but operating conditions were difficult due to the wide inflation and deflation movements of the lung. Without knowledge of the oxygen content or pCO_2 at the time of operation the anaesthetist felt that the patient would become seriously hypercarbic and hypoxic if the pressure of inflation was reduced.

Fluids during surgery: 4000 ml whole blood; 250 ml N/S and 500 ml 5%D/W.
Supportive therapy: High humidity oxygen tent for 16 hours postoperatively.
Uneventful postanaesthetic course.

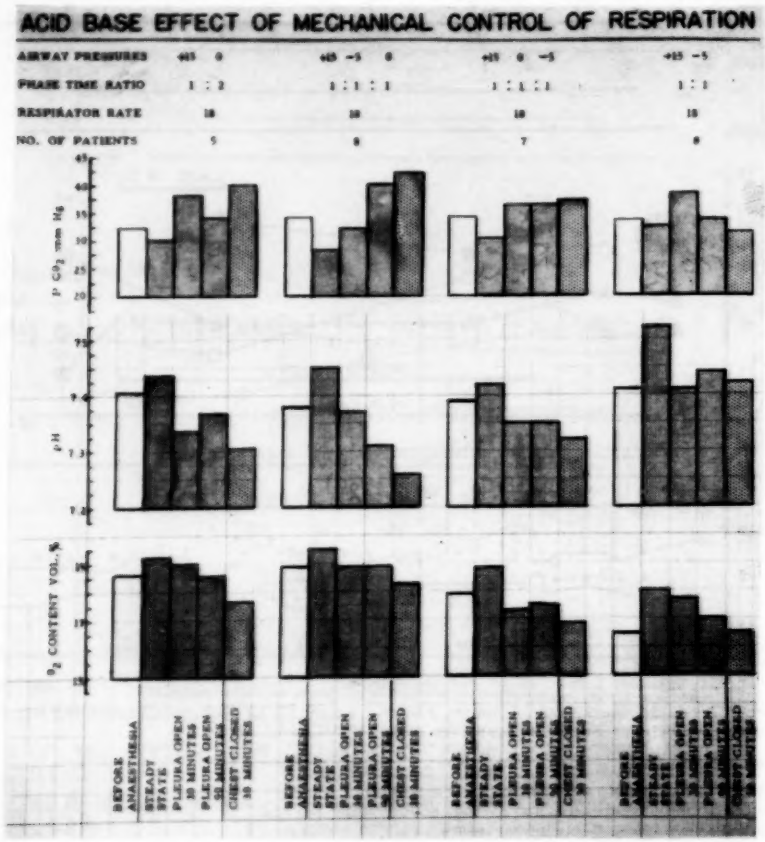

FIG. 9

Bargraph.—Acid-base effect of mechanical control of respiration.

to maintain a normal pH, especially if a large quantity of blood is required for replacement, since freshly stored bank blood has a pH of 6.50 to 6.85 (DeGowin et al., 1949). The maintenance of normal oxygen content is also no simple task, and probably requires that a minimum of 50 per cent oxygen should be administered at all times during intrathoracic anaesthesia and during the first few hours postoperatively.

SUMMARY

During these studies, the anaesthetist, the surgeon, the general type of operation, the patient's disease state, and the anaesthetic method and technique were constant within clinical limits. Amplitude of the breathing curve where a subatmospheric phase was added also was held constant, as laboratory and clinical experience showed the pressure used was most adequate. The variables were restricted to

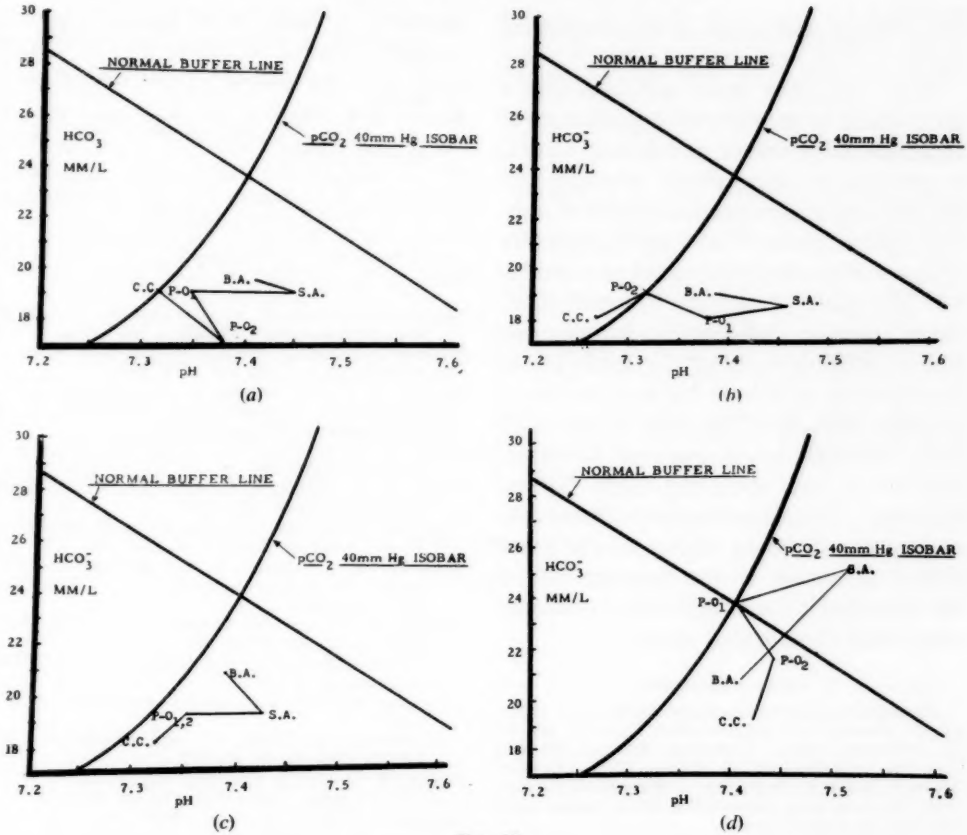

FIG. 10

(*a*) Represents bicarbonate content and pH changes in the 5 patients managed with controlled respiration with airway pressures +15mm Hg; atmospheric pressure; phase-time ratio 1:2; respirator rate at 18/min.

Note. pCO_2 was lowered after steady anaesthesia (SA); increased when pleura was opened; with progress of the operation returned to near normal level; then greatly increased when chest was closed. From before anaesthesia (BA) to steady anaesthesia (SA) bicarbonate fell slightly while pH rose. Both dropped when the pleura was open (PO_1 and PO_2). pH continued to fall after the chest was closed (CC).

(*b*) Represents bicarbonate content and pH changes in the 8 patients managed with controlled respiration with airway pressures +15 mm Hg, −5mm Hg and atmospheric pressure; phase-time ratio 1:1:1; respirator rate at 18/min.

Note. pCO_2 rose slowly and pH fell slowly, while bicarbonate remained steady throughout.

(*c*) Represents bicarbonate content and pH changes in 7 patients managed with controlled respiration with airway pressures +15 mm Hg, atmospheric pressure, −5 mm Hg; phase-time ratio 1:1:1; respirator rate at 18/min.

Note. pCO_2 remained relatively stable throughout. From BA to SA there was slight rise in pH. When pleura was opened there was moderate fall in bicarbonate and pH. This remained quite steady, until the chest was closed (CC), which was followed by a further lowering of bicarbonate and pH.

(*d*) Represents bicarbonate content and pH changes in 8 patients managed with controlled respiration with airway pressures +15mm Hg, −5mm Hg; phase-time ratio 1:1; respirator rate at 18/min.

Note. pCO_2 remained below 40 mm Hg throughout period of anaesthesia while bicarbonate and pH remained at near normal levels throughout the operation.

the position and shape of the breathing curve.

This controlled study indicates that a three-phase respiration cycle consisting of an accelerating inflow to a pressure peak, a rapid fall to atmospheric pressure and the addition of a subsequent phase of subatmospheric pressure is of great assistance in cases where high pressures are needed for adequate pulmonary ventilation, where passive deflation is faulty, and where difficulty is expected in maintaining near-normal acid-base balance and oxygenation because of the type of operation (lung excision) or because of abnormal posture on the operating table (lateral or prone). The subatmospheric phase does not appear to be of added benefit or of added advantage in the management of the anaesthetic under operative conditions other than those stated above.

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(To be continued)

Brit. J. Anaesth. (1956), **28**, 324

AN ELECTRO-ENCEPHALOGRAPHIC STUDY OF THE ANALEPTIC ACTION OF ββ ETHYL METHYL GLUTARIMIDE (MEGIMIDE)

I. Against several Barbiturates and other Hypnotics
II. Compared with other Analeptics and Convulsants

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ββ ETHYL methyl glutarimide (Megimide) was first synthesized in 1911. Its anti-barbiturate activity was first observed by Shaw and others in 1951. It was subsequently investigated by him, in animals (Shaw et al., 1954) and in man (Shulman et al., 1955).

Megimide is of particular clinical interest because attempted suicide and accidental overdose with barbiturates are common, and an effective antidote would help to reduce the morbidity and mortality which results. Such an antidote must be free from dangerous side actions, e.g. convulsions, and effective because careful conservative care without the use of drugs has already brought the mortality down to 1.6 per cent (Clemmensen, 1954) in Scandinavian centres devoted to this problem.

A further clinical application of an antidote is the reversal of barbiturate anaesthesia in outpatients who have had minor surgical procedures. Finally, following neurosurgery performed during anaesthesia maintained by thiopentone, it is desirable to have a quick return to consciousness as variations in the level of consciousness assist in assessing the post-operative course.

Some preliminary electro-encephalographical work was done by the author previously (Shaw et al., 1954) and this work is an extension of it.

EXPERIMENTAL PROCEDURE

Rabbits were used exclusively. No particular type was chosen but most animals weighed about 2 kg. They were kept in equable surroundings, $70 \pm 2^{\circ}$F, and fed on a standard diet (Bruce and Parkes No. 18).

Skin electrodes were used, the hair being shaved and the skin cleaned with acetone. Cambridge electrode jelly was rubbed into the skin and a little placed between the disc electrode and the skin. The electrode was fixed to the skin by celloidin. The use of these electrodes is recommended by the makers of the Grass electro-encephalograph which was used. The resistance between electrodes was repeatedly measured and values below 10,000 ohms were always obtained.

The Grass electro-encephalograph used is a four-channel instrument and proved

absolutely reliable throughout the experiments.

Recordings were made from frontal and occipital electrodes throughout, an indifferent electrode being attached to the right back leg. Fronto-occipital electrodes provide an adequate record of cortical activity in the rabbit and overcome the problem of the limited space which is provided by the small head and large ears.

The animals were allowed to feed and drink normally up to the commencement of the experiment in order to avoid hypoglycaemia or dehydration, and oxygenation was carefully maintained during the respiratory depression caused by the barbiturates. In this way it was hoped to reduce frequency changes, e.g. loss of fast rhythm, which may follow these changes.

The animals were placed in a box to prevent their moving about with subsequent displacement of electrodes. A waking E.E.G. was taken and then the hypnotic drug given by intravenous injection through the marginal ear vein. When hypnosis was sufficiently deep and stable, a further tracing was recorded, and then the analeptic drug was given by intravenous injection. Subsequent to this, tracings were made from control animals who had been given hypnotics but no analeptics.

In this manner it was possible to determine the analeptic effects of Megimide on several barbiturates and other hypnotics, and then to compare the analeptic effect of Megimide with that of other analeptics and convulsants on a rabbit anaesthetized with pentobarbitone. Repeated experiments were performed and the results recorded in this paper are representative of those obtained.

EXPERIMENTS

(1) *Pentobarbitone and Megimide.*

40 mg/kg of pentobarbitone were given after a waking record (fig. 1A). Deep anaesthesia resulted (fig. 1B). This tracing has some alternating current interference, but shows a marked loss of fast frequencies and high voltage seen in the waking record. 20 mg/kg of Megimide were then given, and produced a considerable increase both in fast activity and voltage (fig. 1C).

For comparison with figure 1C, figure 1D is a control taken at a similar time interval in an animal who has had 40 mg/kg of pentobarbitone but no Megimide. This trace shows the very slight electrical activity interrupted by periodic "bursts" characteristic of deep barbiturate hypnosis.

(2) *Thiopentone and Megimide.*

40 mg/kg of thiopentone was given after a waking trace was taken (fig. 2A). This produced a very deep anaesthetic pattern (fig. 2B) with little activity and small bursts of slow waves.

20 mg/kg of Megimide restored the pattern to that of a considerably lighter depth of anaesthesia (fig. 2C) with steady slow waves and some faster waves.

For comparison, figure 2D is taken at a similar time interval after 40 mg/kg of thiopentone without Megimide. It shows less activity both in slow and fast waves.

(3) *Amylobarbitone and Megimide.*

60 mg/kg of amylobarbitone was given after a waking trace was taken (fig. 3A). A subsequent tracing (fig. 3B) shows profound depression of electrical activity with occasional spindles of slow waves.

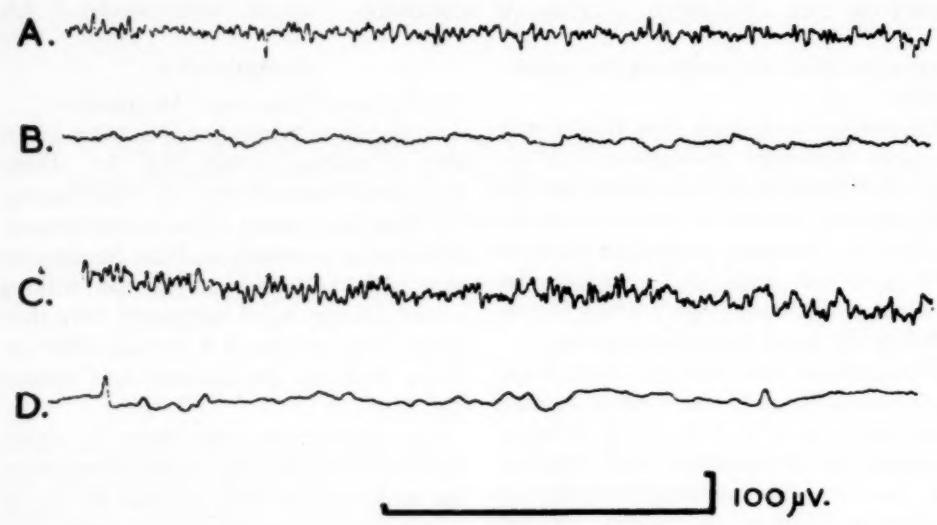


FIG. 1

(A) Awake.
(B) Pentobarbitone (Nembutal) 40 mg/kg.
(C) 5 minutes after (B). Megamide 20 mg/kg.
(D) Control. 5 minutes after (B). No Megimide given.

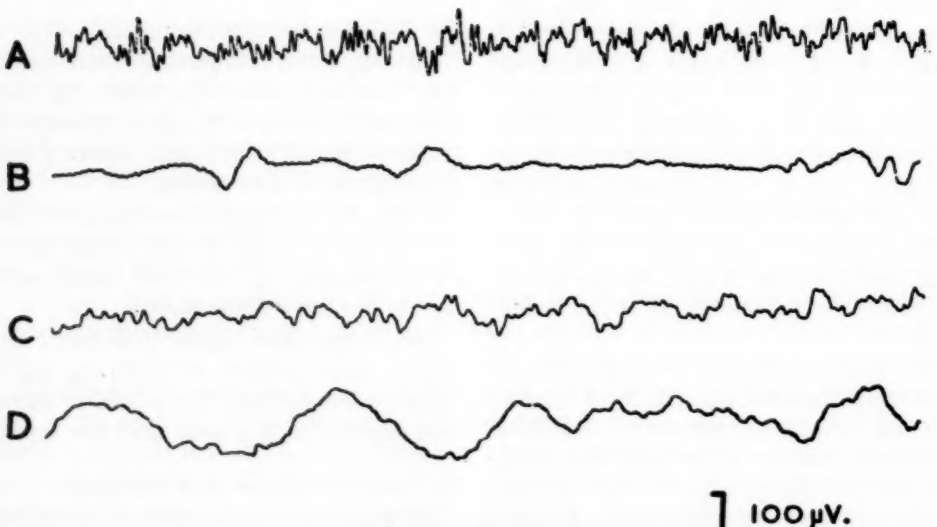


FIG. 2

(A) Awake.
(B) Thiopentone 40 mg/kg.
(C) 8 minutes after (B). Megimide 20 mg/kg.
(D) Control. 8 minutes after (B). No Megimide given.

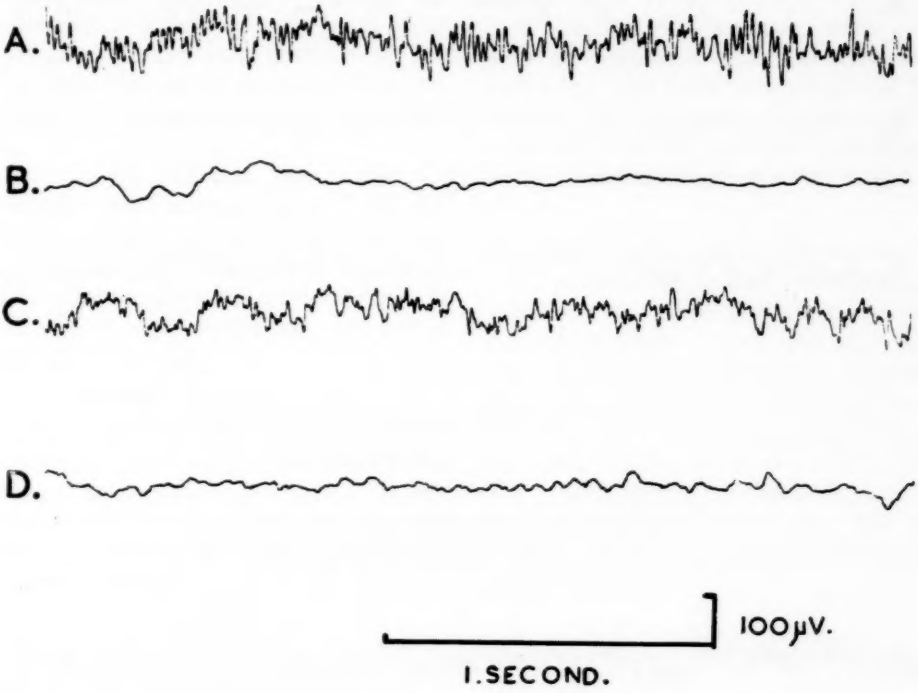


FIG. 3

(A) Awake.
(B) Amylobarbitone (Amytal) 60 mg/kg.
(C) 14 minutes after (B). Megimide 10 mg/kg.
(D) Control. 14 minutes after (B). No Megimide given.

10 mg/kg of Megimide restored the tracing to an appearance (fig. 3C) approaching the waking pattern, the main difference being in the increased number of slow waves and the lower voltage. These are typical of the lightest stages of hypnosis with barbiturates.

Figure 3D is a tracing taken at a much later time from an animal who had 60 mg/kg of amylobarbitone without Megimide. It shows only moderately slow waves at a low voltage and indicates a considerably greater depth of depression than figure 3C.

(4) *Thialbarbitone (Kemithal) and Megimide.*

A waking tracing was made (fig. 4A) and thialbarbitone 200 mg/kg given. This produced a tracing (fig. 4B) with very little activity apart from spindles of slow waves. (This tracing has regular electrocardiographic artifacts.)

20 mg/kg of Megimide restored much fast activity (fig. 4C) and increased the voltage of the slow waves, thus restoring the typical wave pattern of light barbiturate hypnosis.

(5) *Chloralose and Megimide.*

After a preliminary tracing (fig. 5A) 70 mg/kg of chloralose was given. A tracing (fig. 5B) was obtained with high voltage slow delta waves (about 2 per second) predominating.

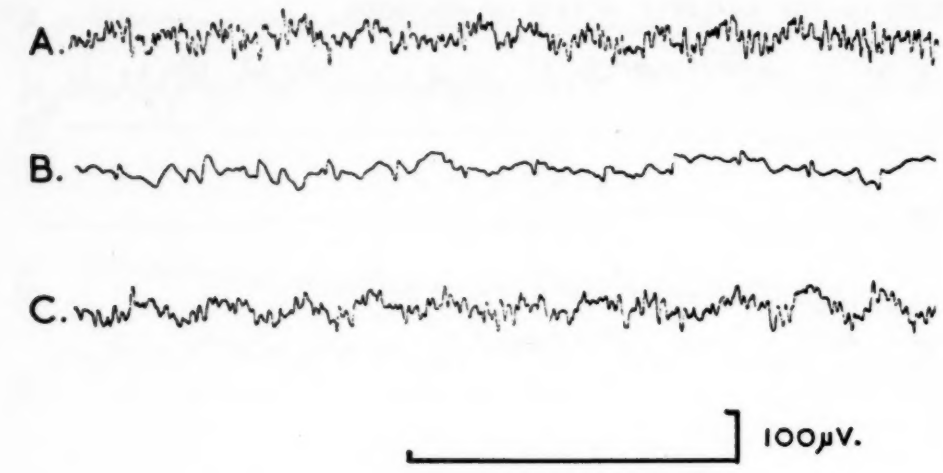


FIG. 4

(A) Awake.
(B) Thialbarbitone (Kemithal) 200 mg/kg (E.C.G. complexes produce regular artifacts on this record).
(C) 6 minutes after (B). Megimide 20 mg/kg.

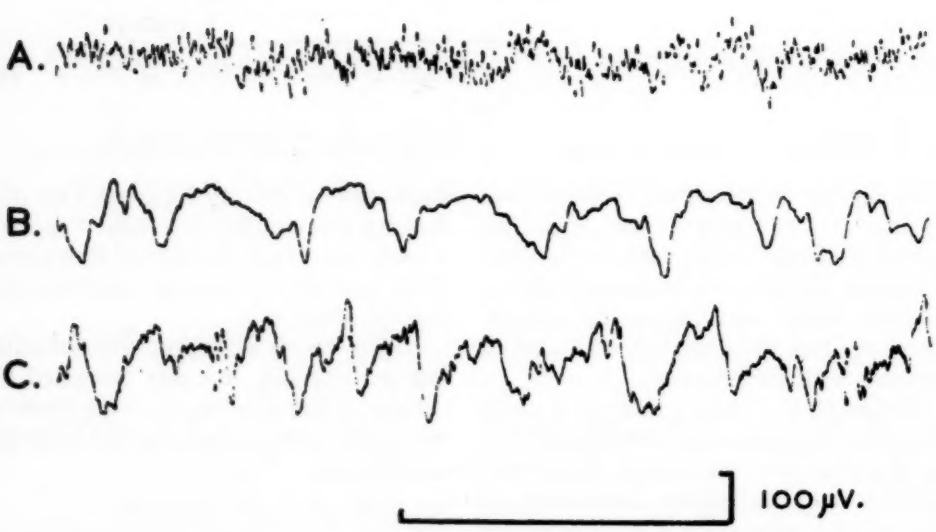


FIG. 5

(A) Awake.
(B) Chloralose 70 mg/kg.
(C) 5 minutes after (B). Megimide 5 mg/kg.

Megimide 5 mg/kg was given when some faster β activity was restored (fig. 5C). The delta frequency persisted, however, and the rabbit showed no signs of awakening.

It was noticed that the rabbit was twitching (see electromyogram in fig. 5C) and very restless after the Megimide, suggesting that chloralose did not antagonize the convulsant property of the drug. The restlessness was controlled by pentobarbitone 5 mg/kg.

(6) *Diethyl Ether and Megimide.*

After a preliminary tracing (fig. 6A) a rabbit was given an ether/air anaesthetic. When a deep plane was reached and the intercostals were paralysed, figure 6B was obtained, showing the characteristic low-frequency activity.

10 mg/kg of Megimide were given when an increase in electrical activity resulted (fig. 6C) both in voltage and frequency. This increased activity is small compared with the response to Megimide during barbiturate anaesthesia (cf. figs. 1B and 1C).

(7) *Methylpentynol and Megimide.*

A 5 per cent solution of methylpentynol (Oblivon) was given, 250 mg/kg producing a deep stage of sleep (fig. 7B) with a very sluggish corneal reflex. Repeated small doses of the drug were necessary to maintain this stage, suggesting that redistribution from the blood was occurring. The record shows very slow frequencies (1–2 per second) and very little fast activity. There is a regular electrocardiographic artifact.

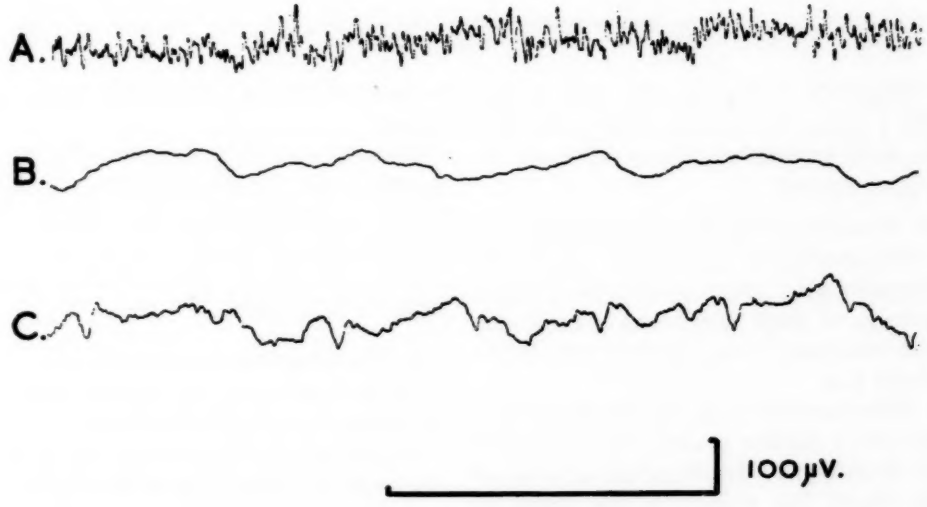


FIG. 6

(A) Awake.
(B) Ether anaesthesia (3rd plane).
(C) 3 minutes after (B). Megimide 10 mg/kg.

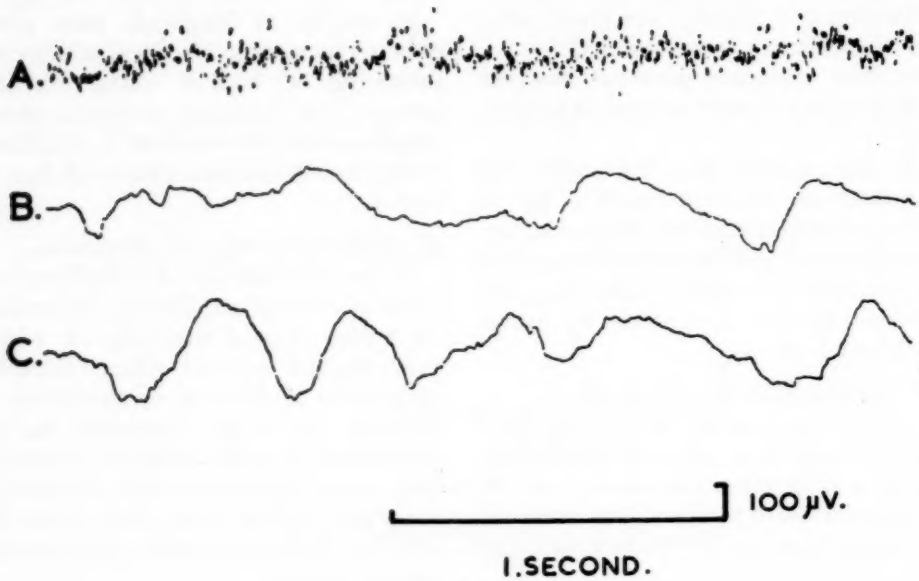


FIG. 7

(A) Awake.
(B) Methylpentynol (Oblivon) 250 mg/kg.
(C) 1 minute after (B). Megimide 5 mg/kg.

Megimide 5 mg/kg was then given. This produced a minimal change (fig. 7C), the slow frequencies being increased to 2–3 per second.

(8) *Pentobarbitone and Nikethamide.*

Pentobarbitone was given until the characteristic electro-encephalographic features of deep barbiturate anaesthesia were obtained; 55 mg/kg were required to obtain this.

Nikethamide 75 mg/kg was then injected and a further tracing (fig. 7C) shows no alteration in the electrical activity of the brain. No change in the depth of anaesthesia was observed directly.

(9) *Pentobarbitone and Methylamphetamine.*

Deep anaesthesia (fig. 8B) was obtained by pentobarbitone, 40 mg/kg being required.

Subsequent administration of methylamphetamine (Methedrine) 2 mg/kg produced a minimal electrical change (fig. 8C) consisting of bursts of about 18 cycles per second, and no visible change in the depth of anaesthesia.

(10) *Pentobarbitone and Picrotoxin.*

Deep anaesthesia (fig. 9B) was obtained by 40 mg/kg of pentobarbitone.

Picrotoxin in a dose of 0.5 mg/kg produced an increase in both frequency and amplitude of the electrical waves (fig. 9C). This indicates a small reduction in depth of anaesthesia, but there is no fast β activity such as is obtained by Megimide (compare fig. 1C).

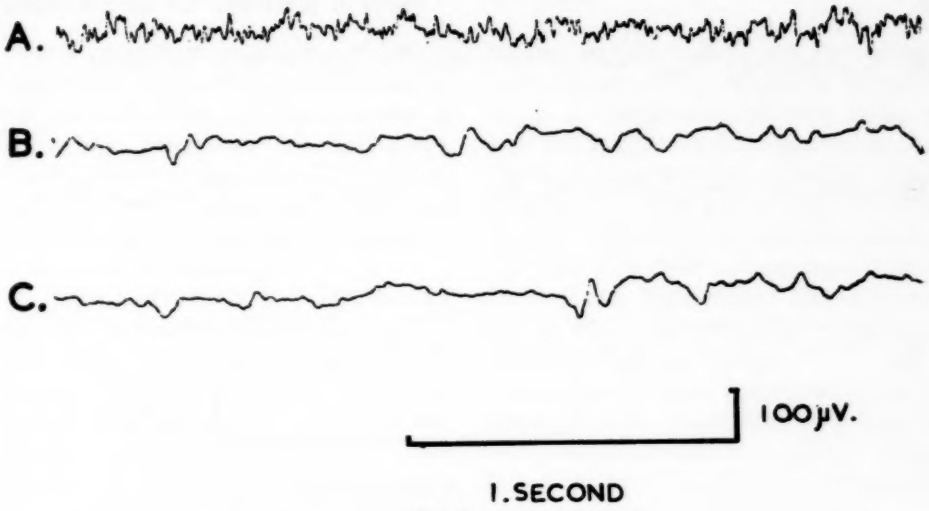


FIG. 8

(A) Awake.
(B) Pentobarbitone (Nembutal) 40 mg/kg.
(C) 6 minutes after (B). Nikethamide (Coramine) 75 mg/kg.

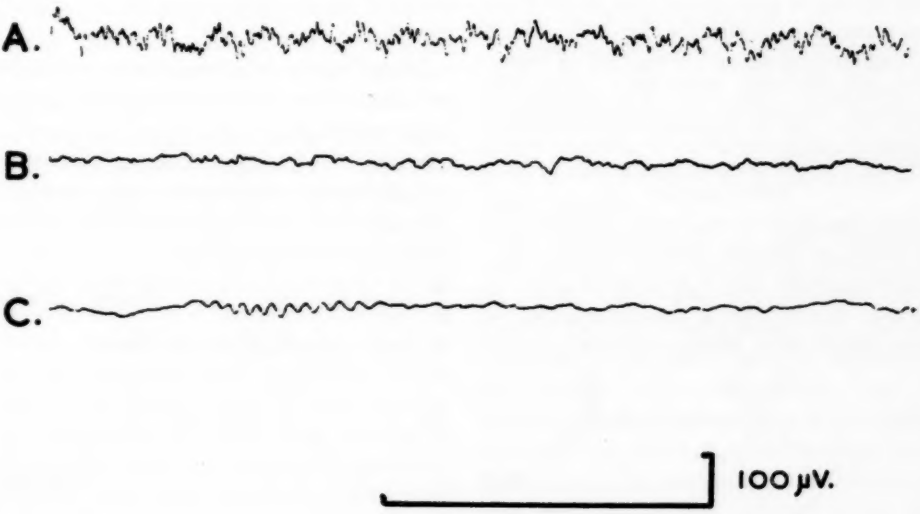


FIG. 9

(A) Awake.
(B) Pentobarbitone (Nembutal) 40 mg/kg.
(C) 5 minutes after (B). Methylamphetamine (Methedrine) 2 mg/kg

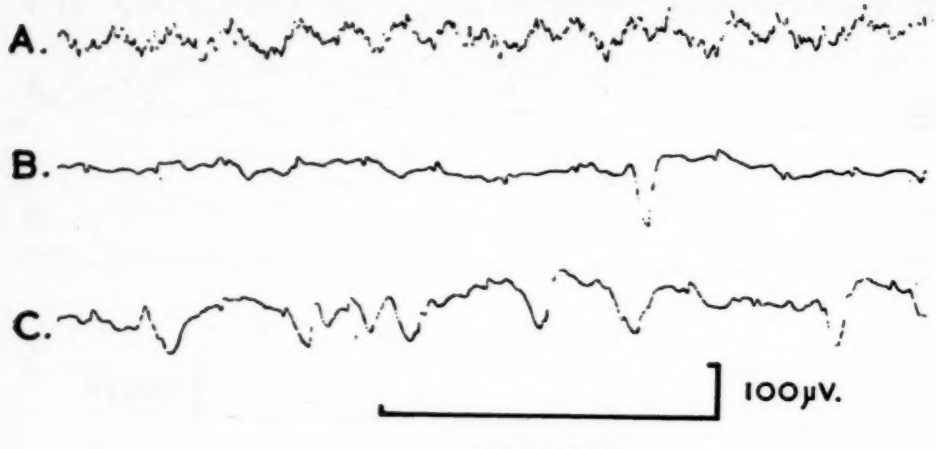


FIG. 10

(A) Awake.
(B) Pentobarbitone (Nembutal) 40 mg/kg.
(C) 10 minutes after (B). Picrotoxin 0.5 mg/kg.

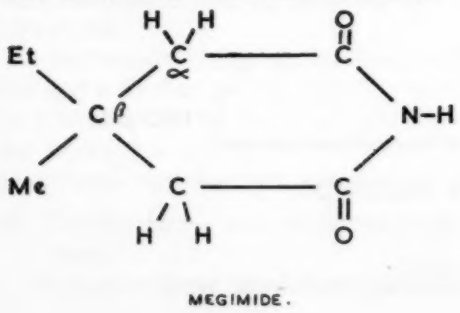
(11) *Pentobarbitone and Leptazol.*

40 mg/kg of pentobarbitone produced a deep stage of anaesthesia (fig. 10B) which was unchanged after 7.5 mg/kg of leptazol were given (fig. 10C). This tracing in figure 10C has a regular electrocardiographic artifact.

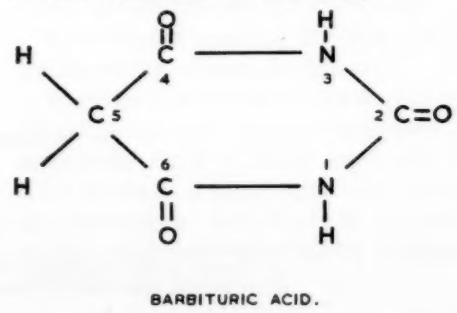
DISCUSSION

The biophysical processes resulting in the electrical changes shown in the electroencephalograph are not fully known at present. In this work the assumption is made that a drug which restores an electrical pattern similar to that of light anaesthesia is antagonizing the anaesthetic used.

There are several types of antagonism possible but the evidence obtained in these experiments does not suggest any particular type although Megimide shows specificity in antagonizing only barbiturates. There is a similarity in part of their molecules, the grouping $\begin{matrix} R \\ R \end{matrix}\!\!>\!C\text{—}CO\text{—}NR\text{—}CO$ being common to both (Marshall et al., 1954).



MEGIMIDE.



BARBITURIC ACID.

It is interesting to note that the α phenyl glutarimides resemble the barbiturates in their anticonvulsive action, whereas ββ ethyl methyl glutarimide is a convulsant. Its convulsant actions are readily opposed by small doses of barbiturates, and it can in turn shorten the duration of sleep produced by the barbiturates.

A further observation of interest has been made during preliminary experiments with α ethyl α phenyl glutarimide (Doriden, Ciba). It was found that sleep induced by this agent in mice was antagonized rapidly by Megimide. The similarity of structure of the two molecules suggest that competitive inhibition may account for this.

The existence of convulsive and anticonvulsive compounds with the same nucleus is seen also in the derivatives of barbituric acid. Powell et al. (1943) discuss this and describe a compound which is convulsant in warm-blooded animals and depressant in cold-blooded.

The effects of depressant and excitant drugs and anaesthetics on the electroencephalogram have been described by Gibbs and Maltby (1943) and Gastaut et al. (1951). Their results were reproduced in these experiments and were used in the interpretation of the changes observed. In general, anaesthetics and depressants reduce the frequency and amplitude of the wave traces. Other effects, such as the high amplitude and very low frequency theta waves seen in figure 5B, are mentioned as they occur in the experimental data.

CONCLUSIONS

In figure 1 it can be seen that Megimide (20 mg/kg) given after pentobarbitone (40 mg/kg) restores the trace to a nearly normal pattern (1C), while the animal to whom no Megimide has been given retains the pattern of deep depression (1D). Figure 2 compares the activities of two animals who were given thiopentone (40 mg/kg). The pattern of light depression of (2C) after Megimide (20 mg/kg) contrasts the very slow high-voltage theta waves of (2D) in which no antagonist was used and deep depression persists.

A similar reversal of pattern is shown in figure 3C by Megimide (10 mg/kg) after amylobarbitone (60 mg/kg). Figure 3D is a control tracing with persistent slow waves at a lower voltage.

The response to Megimide (20 mg/kg) following thialbarbitone (Kemithal), 200 mg/kg, shown in figures 4B and 4C, again suggests a lightening of the depression due to this thiobarbiturate.

From these results, therefore, it can be seen that Megimide reverses the e.e.g. changes induced by commonly used barbiturates and thiobarbiturates. Attention is therefore directed to some nonbarbiturate hypnotics.

A typical pattern of deep chloralose depression (70 mg/kg) is shown in figure 5B, with very slow high-voltage waves predominating. Megimide (5 mg/kg) increases the activity (fig. 5C), some faster low-voltage waves appearing, but the dominant pattern is still slow high-voltage waves. There was no sign of waking in the rabbit and it is concluded that Megimide does not antagonize chloralose to any extent. The appearance of muscle twitching is interesting as it suggests that chloralose does not antagonize Megimide as do the barbiturates.

Ether anaesthesia was induced and a deep plane obtained (fig. 6B). Activity in this tracing is minimal. Megimide (5mg/kg) increased both fast and slow waves (fig. 6C) but this improvement is small compared with that typically seen with the barbiturates (figs. 1B and 1C).

Methylpentynol (250 mg/kg) produced a tracing (fig. 7B) in which very little fast activity is seen and the slow high-voltage waves seen with deep chloralose depression are dominant. Megimide (10 mg/kg) had scarcely any effect on the pattern of activity (fig. 7C).

It was concluded from the observations that Megimide produced minimal change in animals depressed by nonbarbiturate hypnotics. These changes are probably caused by a nonspecific physiological antagonism due to the convulsant action of the drug and can be produced by other drugs stimulating the central nervous system (e.g. leptazol).

The more pronounced reversal of activity which Megimide produces after barbiturates may indicate a more specific pharmacological antagonism to them. To investigate this it is proposed to synthesize a nonconvulsant form of Megimide by substituting an α phenyl group on it. This alteration has been shown to produce anticonvulsant activity in other glutarimides (Marshall et al., 1954).

Megimide was next compared with other commonly used analeptics and convulsants. In each case pentobarbitone was given until a pattern of deep depression was obtained (figs. 8B, 9B, 10B, 11B). Nikethamide (75 mg/kg) produced virtually no change (fig. 8C). Methylamphe-

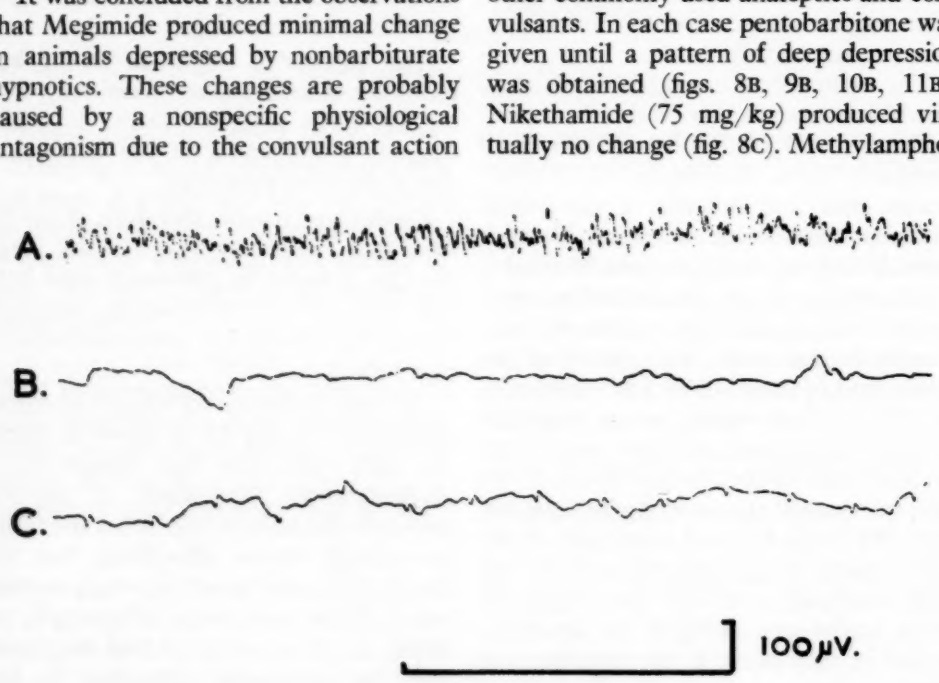


FIG. 11

(A) Awake.
(B) Pentobarbitone (Nembutal) 40 mg/kg.
(C) 15 minutes after (B). Leptazol 7.5 mg/kg.

tamine (2 mg/kg) was followed by regular, even bursts of low-voltage waves (18 cycles/second) but no further evidence (fig. 9c) of lessened depression.

Picrotoxin (0.5 mg/kg) resulted in a greater return of activity (fig. 10c) than the former analeptics. The pattern is predominantly slow, however, and does not compare with that following Megimide (fig. 1c).

The convulsant drug leptazol was given in a dose of 7.5 mg/kg. Virtually no change in the tracing resulted (fig. 11c). Allowance must be made in this record for the regular electrocardiographic complexes.

It is concluded from these observations that Megimide is a more effective barbiturate antagonist than the commonly used analeptics. Its chief disadvantage is its convulsant action when given to patients who have not had barbiturates previously. The same difficulty accompanies the use of picrotoxin.

This activity is very readily suppressed by very small doses of barbiturate given intravenously. As mentioned previously, an attempt is being made to reduce the convulsant activity by slight alteration to the drug.

SUMMARY

An electro-encephalographic study of Megimide has demonstrated the following:

(1) It markedly reverses the pattern of deep depression due to all barbiturates and thiobarbiturates tested.

(2) It does not reverse the pattern of deep depression due to some nonbarbiturate hypnotics and anaesthetics.

(3) It is capable of reversing the deep barbiturate pattern more than the commonly used analeptics and convulsants.

ACKNOWLEDGMENTS

I wish to thank Professor D. Slome and Dr. B. D. Wyke for their interest in this work. My grateful thanks are due to Mr. C. H. Redman, A.I.S.T., for his able technical assistance during the experiments and in preparing part of this paper. Nicholas Products Laboratories Limited supplied generous amounts of Megimide; Ciba Laboratories supplied the Doriden.

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EDITORIAL ANNOUNCEMENT

It is recognized that the subscription of £3 15*s*. per annum for the *British Journal of Anaesthesia* might be a heavy burden for anaesthetists in trainee positions, despite the fact that for this subscription they receive twelve monthly issues, including two of special educational interest. Because of our desire to interest aspirants to the specialty in this publication, the publishers have agreed to supply the Journal to *bona fide* trainees in Great Britain, on application for three years at the reduced rate of £2 10*s*.

Forms of application for this concession may be had on request to the Editors.

Brit. J. Anaesth. (1956), **28**, 336

CARDIAC ARREST FOLLOWING THE ADMINISTRATION OF NEOSTIGMINE

BY

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Maryfield Hospital, Dundee

As the vagal stimulation of neostigmine is well known it is customary in anaesthetic practice to inject this drug with an antagonizing dose of atropine. The atropine may precede the neostigmine by two or three minutes or the two drugs may be given together, though the danger of this procedure is recognized (Bain and Broadbent, 1949). The following is a description of a case in which cardiac arrest followed the simultaneous injection of these drugs.

The patient, a well conditioned man of 73 years, was suffering from a cancer of the rectum for which an abdominal perineal resection was proposed. There was no evidence of myocardial weakness, his pulse was regular, the rate being 72 per minute and his resting blood pressure 160/80 mm Hg.

Premedication consisted of morphine 1/6 grain (10 mg) and atropine 1/100 grain (0.6 mg) one and a half hours before operation, and to diminish blood loss it was decided to combine spinal analgesia with a light general anaesthetic.

At 3.15 p.m. with the patient in a sitting position lumbar puncture was performed in the third lumbar interspace. Heavy cinchocaine 0.75 ml followed by light cinchocaine 13 ml were injected. Fifty seconds later the patient was placed on his back and the table given a ten degrees head down tilt. At 3.30 p.m. thiopentone 400 mg and gallamine 120 mg were given intravenously and intubation accomplished with a size nine cuffed endotracheal tube. Respiration was now controlled and anaesthesia continued by inflation with nitrous oxide two parts and oxygen one part with carbon dioxide absorption. The systolic blood pressure was 70 mm Hg and the pulse slow and regular.

With the patient now in a head down lithotomy position the operation was begun. At 4.10 p.m. a blood drip was set up and at 4.15 p.m. pethidine 50 mg was injected. Spontaneous respiration was by this time returning, so for the the next three-quarters of an hour respiration was assisted while the clinical picture remained unchanged—a quiet pink patient with contracted pupils, a slow palpable radial pulse, and the small blood loss adequately replaced. The operation was completed at 5.05 p.m., no cause for anxiety to surgeon or anaesthetist having occurred.

Breathing now appeared adequate but to neutralize any remaining relaxant, neostigmine 2.5 mg mixed with atropine 1/100 grain (0.6 mg) were injected intravenously, the patient still being in the lithotomy position, and the table still with a head down tilt. Almost at once he collapsed, the complexion became grey, the pupils widely dilated and the peripheral pulse nowhere palpable.

The abdominal wound was immediately opened and the heart was found to have stopped. The surgeon carried out cardiac massage in the classical manner but without incising the diaphragm, while the lungs were kept inflated with oxygen. Methylamphetamine 30 mg was injected into the drip and nikethamide 2 ml was injected into the heart. As there was no response to these measures a long wide bore needle was then inserted into the heart in the fourth intercostal space 2½ inches (6.25 cm), from the mid sternal line on the left side. Through this needle blood was forced into the heart at a rapid rate, 500 ml being given in three minutes. When about 300 ml had been injected the heart was felt to give some feeble contractions, and within a matter of seconds it was beating vigorously. In a like manner respiration returned, shallow at first but followed in a few moments by deep exaggerated breathing. Five minutes later pulse and respiration were satisfactory and the systolic blood pressure was 110 mm Hg.

At 5.45 p.m. with reflex activity returning but still tolerating an oral airway the patient was returned to the ward. He remained unconscious for about five hours until at 11 p.m. he asked feebly for a drink. Next morning, sixteen hours after the cardiac arrest, he was awake and alert, there was no disorientation and his speech and behaviour were normal.

DISCUSSION

The danger of giving neostigmine mixed with atropine is attributed to the transient vagal stimulation of the atropine coinciding with the vagal stimulation of the neostigmine. Deaths have been attributed to this procedure (Macintosh, 1949) and it has been suggested (Clutton-

Brock, 1949) that not only should the drugs be injected separately but that the neostigmine should be given in divided doses.

In this case there was the additional hazard of the sympathetic paralysis following the spinal anaesthetic so it seems reasonable to suppose that the vagus would have a very free rein and that any vagal stimulation would have a manifold effect.

Cardiac arrest must have lasted at least five minutes, and it is of interest that contractions did not return with cardiac massage, but commenced dramatically as soon as the heart was filled with blood. Iokhveds (1944) has described the value of intracardiac transfusion in several cases, and in this case I was impressed by its efficacy. It is understood that analeptic drugs injected into a stagnant circulation would be valueless, but once the heart started to beat they may have had some effect.

ACKNOWLEDGMENTS

I must express my appreciation to the surgeons, Mr. J. S. Kinnear and Mr. W. Walker, for their prompt and energetic handling of the situation, and to those present in the theatre for their co-operation in the emergency.

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BOOK REVIEWS

Stellate Ganglion Block. By Daniel C. Moore, M.D., Director, Department of Anaesthesiology, Mason Clinic, Seattle, Washington. Published by Charles C. Thomas, Springfield, Ill., U.S.A. Pp. 280; 101 illustrations.

Complications of Regional Anaesthesia. By Daniel C. Moore, M.D. Published by Blackwell Scientific Publications, Oxford. Pp. 291; 51 illustrations. Price 75*s*.

Dr. Moore has achieved a considerable reputation both in this and his own country as an exponent of regional analgesia. These two books maintain if not enhance the high standard he set with his first book on Block Anaesthesia. That a book of some size can be devoted to stellate ganglion block alone is an indication of the exhaustive way that this subject is treated. The anaesthetist, rightly, is not regarded by Dr. Moore as a technician but as a physician who needs to know a good deal about the clinical conditions for which stellate block is performed. The anatomy and physiology of the thoracic and cervical sympathetic nervous system, and the pharmacology of the drugs used are discussed in some detail. Then come descriptions, easily followed and beautifully illustrated, of the methods advised by the author. One of the outstanding values of the book lies in the chapters on clinical indications and uses of stellate block. A very large number of these are described; the reader is likely to have his eyes opened to the possibilities of this block. An appendix is included which describes numerous methods other than those advised by the author.

In the second book Dr. Moore has gathered together from his own vast experience and from the literature a collection of complications which at first sight might frighten the novice from ever attempting local analgesia. These complications range from allergic reactions to oxygen want and from puncture of blood vessels to inadvertent spinal analgesia. However, as he repeatedly points out, these complications are rare with ordinary precautions. As a reference book it will not be surpassed, for it is doubtful if any more comprehensive survey of these complications could be made.

These two books are massive contributions to an already large literature on local analgesia. They are up to date and are largely based on a wide personal experience. They are well written and profusely illustrated, and they are almost certainly the most important recent additions to the literature on this subject.

W. W. Mushin

Brit. J. Anaesth. (1956), **28**, 338

MODIFIED ROUTINE SUXAMETHONIUM INFUSION

BY

R. C. OLIVA

Connaught Hospital, London

THE administration of suxamethonium chloride (Scoline) in anaesthesia through the medium of an intravenous saline infusion together with a high concentration of oxygen in a nitrous oxide-oxygen mixture is so satisfactory that it is used as routine throughout some operating sessions in many hospitals.

The following simple method results in one "giving-set" being used throughout the whole operating session.

Apparatus used.

(1) A glass connection with 2½-inch long rubber tubing attached to each end (diameter of rubber tubing same as giving-set tubing).

(2) Two Luer-Record adaptors.

(3) A Guest canula (figs. 1, 2).

(4) A Y glass connection is used if the suxamethonium infusion and blood transfusion are administered simultaneously (fig. 3).

Technique.

A transfusion giving-set is connected to the suxamethonium solution for infusion. The male end of the Luer fitting adaptor at the end of tubing is wrapped into an alcohol soaked swab and secured with an artery forceps.

A Guest canula is introduced into a suitable forearm vein and strapped in position. The patient is anaesthetized and intubated. The sterile glass connection-tubing set is connected to the "giving-set" and air is expelled by holding it upright and allowing the suxamethonium infusion

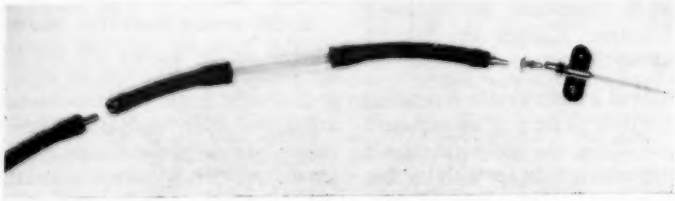

FIG. 1

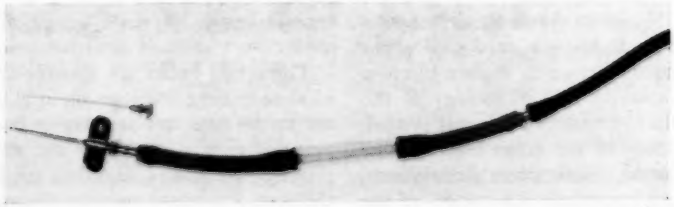

FIG. 2

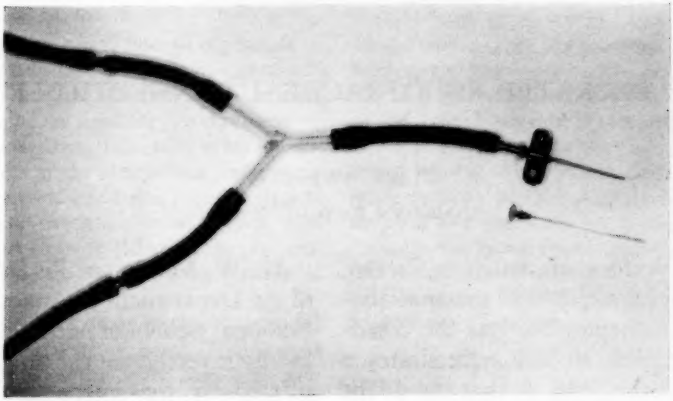

FIG. 3

to run slowly. The set is then connected to the Guest canula.

At the end of the operation the "giving-set" is disconnected, the male Luer adaptor of the "giving-set" is wrapped into an alcohol soaked swab and secured by artery forceps, and so is prepared for the next case.

If the Y-shaped glass connection is used, one end of the Y-connection tubing is connected to the suxamethonium infusion, the other to the blood transfusion and the stem to the canula. The Y glass connection is first filled with suxamethonium infusion before blood is run. The blood bottle should be on a lower level than the suxamethonium infusion.

The glass connection set is used to make certain that the patient's blood does not contaminate the "giving-set" tubing of the suxamethonium infusion. If no blood is seen in the upper third of the glass connection, one can be reasonably certain that the "giving-set" tubing is not contaminated.

Summary.

A method of administering a routine suxamethonium infusion is described permitting the use of only one "giving-set" for a whole operating session.

Simultaneous use of a suxamethonium infusion and a blood transfusion is made possible by the use of a Y-shaped glass connection.

ACKNOWLEDGMENTS

I am indebted to Dr. L. H. Lerman, Consultant Anaesthetist to the Connaught Hospital, for his encouragement, and I also wish to thank Mr. J. Webster, Chief Technician, Pathological Department, Connaught Hospital, for his help with photographs.

Brit. J. Anaesth. (1956), **28**, 340

A PERFORATED ENDOTRACHEAL CONNECTION PIECE

BY

MARK SWERDLOW

Salford Royal Hospital

THE Cardiff perforated connector (Picken, 1950) is a useful addition to the anaesthetist's armamentarium but has the disadvantages (*a*) that its use necessitates a double connection, and (*b*) that should the need to assist the respiration arise then the connector will have to be removed.

I have eliminated these disadvantages in a connection piece illustrated in figure 1.

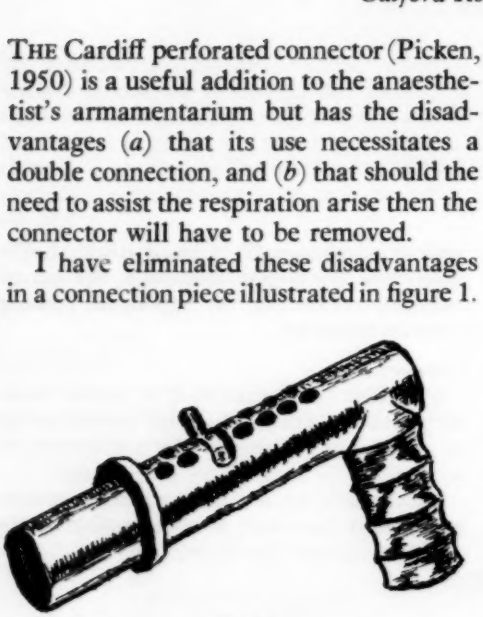

FIG. 1

Basically this is a Rowbotham type connection piece with elongated stem in which the perforations have been made. A rotating inner drum with matching perforations allows partial or complete occlusion of the holes when necessary. The internal diameter of the stem is the same as in a standard Rowbotham piece.

I have used the connection in clinical practice for over twelve months and have found it very satisfactory both in children and adults.

W. W. Mapleson, Ph.D., the physicist of the Department of Anaesthetics, Welsh National School of Medicine, Cardiff, has kindly investigated the physical resistance offered by the instrument (perforations open) and his report is as follows:

Measurements made on 10 *mm bore connection piece*

Flow (litres/min)	Pressure drop across connector (mm H_2O)
10	1
20	3
30	7
40	13

"In inspiration most if not all of the patient's requirements will, it is assumed, be supplied by the fresh gas supply so that the amount of air breathed in through the perforations will be so small as to give a negligible pressure drop across them. As regard expiratory resistance, even with as high a flow rate of fresh gases as 15 litres per minute the connector compares very favourably with any ordinary expiratory valve" (Mushin and Mapleson, 1954).

ACKNOWLEDGMENT

I wish to express my gratitude to Dr. Mapleson for his assistance and to Professor W. W. Mushin, for his advice.

The connection piece is made in different sizes and can be obtained from Medical and Industrial Equipment Ltd., New Cavendish Street, London, W.1.

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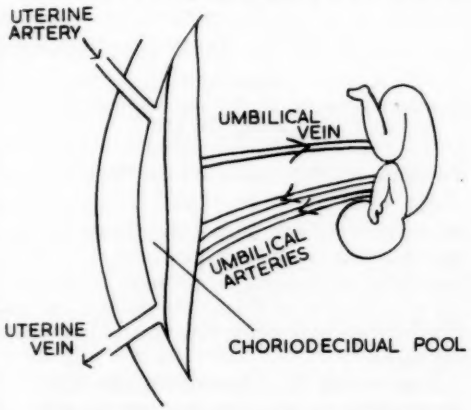
CORRESPONDENCE

SOME ASPECTS OF OBSTETRIC ANAESTHESIA

Sir,—I have read Dr. J. Selwyn Crawford's article (*Brit. J. Anaesth.*, **28**, 146) with interest, but I fear a few points need further comment.

I cannot see how, from the results of his investigations, he arrives at the conclusions in his summary: "The maternal and foetal levels at time of delivery were approximately equal over a wide range of operation times (3–30 min), the placenta, apparently, offering no barrier to the passage of thiopentone."

The article does not state whether the blood samples were arterial or venous. Thiopentone levels in the arteries or the veins cannot be presumed to be equal in either the mother or the foetus. The first "round" of arterial blood after a rapid injection of 250 mg of thiopentone may be expected to have up to 250 μg of thiopentone per ml of blood, even allowing 1 litre for the central blood volume. This means that the choriodecidual space will be subjected to this large concentration of thiopentone (see figure). When the



thiopentone comes round to the peripheral veins, a large quantity will have been removed by the tissues, so that the venous level will give no indication of the concentration to which the placenta is being subjected.

Brown and Veall (1953) found that radio-active isotopes, if injected into the choriodecidual pool, dropped to half strength in 25 seconds in normal pregnancy and in 75 seconds in eclampsia. This would suggest that the maternal arterial blood is held up for some time in this space in pregnancy. There is probably a very much longer delay late in the second stage of labour, especially if there is foetal distress. Thus the placenta may be subjected to a very high concentration of thiopentone while that in a peripheral artery may have already dropped to a low level (i.e. time lag).

Again, the blood vessel (umbilical vein) feeding the foetus with thiopentone cannot be expected to give a true representation of the thiopentone level of the foetal serum.

E. J. O'MULLANE
*St. Thomas's Hospital, London, S.E.*1

REFERENCE

Brown, J. McC., and Veall, N. (1953). *J. Obstet. Gynaec. Brit. Emp.*, **60**, 141.

Sir,—I would like to thank you and Dr. O'Mullane for the courteous way in which I have been given the opportunity of reading his letter prior to publication, thus enabling me to reply in the same issue.

After reading Dr. O'Mullane's criticism, and the article to which he refers, I am happy to say that I find my faith in the conclusions expressed in the article unshaken. It should, of course, have been made plain that the samples of maternal blood were venous. As indicated, the first sample was taken two minutes after the administration of thiopentone, and the crucial sample "B" at periods ranging from three to thirty minutes after administration. I am rather pressed for time at the moment, and have not been able to look up the literature on the subject, but I imagine that after such a period the difference between the thiopentone concentration in the arterial and venous phases of one circulation "round" would be insignificant relative to the actual concentration involved. As far as I have been able to ascertain, all the previous workers in this field have used venous blood to obtain the maternal serum concentration for comparison with the foetal concentration. A similar argument applies to the foetal samples. Blood was obtained from all the contained arterial and venous vessels of the umbilical cord.

With regard to the applied significance of the work of Brown and Veall (1953), these workers

contended that, in normal pregnancy between about the 38th and the 40th weeks, the placental pool volume was approximately 250 ml blood. The maternal placental blood flow is in the order of 600 ml per minute (i.e. about one-tenth of the total cardiac output); it is reduced to about one-third of this figure in pre-eclampsia and in chronic hypertension. It is thus unlikely that the "very high concentration of thiopentone" in the placenta, feared by Dr. O'Mullane, will actually develop.

Furthermore, Brown and Veall indicated that working with No. 24, even when this substance was injected directly into the placental pool, most of the isotope was distributed through the maternal circulation instead of crossing the placental barrier.

Finally, I would point out that in almost all of our cases the maternal and foetal serum thiopentone levels were in actual fact equal, as were, in the smaller series, the bromide concentrations. Surely then, it is not too much to ask that the foetus be regarded as an "extra limb" as far as the distribution of thiopentone administered to the mother is concerned?

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Royal Free Hospital, London

REFERENCE

Brown, J. McC., and Veall, N. (1953). *J. Obstet. Gynaec. Brit. Emp.*, **60**, 141.

DUAL ACTION OF SUXAMETHONIUM CHLORIDE

Sir,—I should like to be among the first to congratulate Dr. H. J. Brennan on his excellent paper "Dual action of suxamethonium chloride" (*Brit. J. Anaesth.*, 1956, **28**, 159). However, the conception which is created—namely that the suxamethonium changes its mode of action—is misleading. I would, therefore, with your permission like to review some of the evidence to date.

In 1952, Dr. Richardson and I described a "dual type of response" occurring under certain conditions in man: at the same time Dr. Zaimus reported a similar finding in some species of animals. Together we developed the thesis that there was, in fact, a new and third type of neuromuscular block. Its principal features were that at first it showed signs of depolarization, but these rapidly changed until it became typical of a non-depolarization block, i.e. like d-tubocurarine. This type of block was named a "dual block" because it emphasized that two salient features were concerned and they occurred in a precise order.

The administration of decamethonium to cases of myasthenia gave rise to a dual block, yet this drug could be recovered unchanged from the urine of these patients and when injected into the chick caused a pure depolarization block (Churchill-Davidson and Richardson, 1953). This evidence suggests that in the dual response it is the *motor endplate* and not the depolarizing drug which changes its response. Dr. Brennan now brings forward evidence that under certain conditions suxamethonium can produce a similar type of block in normal subjects, but in his conclusion states that the drug changes its mode of action. From the fact that he uses our original terminology we must assume that he does not consider the response differs radically from that already described with decamethonium in myasthenia. I think, therefore, it is most important to emphasize that in our conception of the "dual block" it is the response of the motor endplate, and not the drug, which is the principal factor concerned.

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Sir,—I am very sorry indeed that the references I quoted failed to make it clear that Dr. H. C. Churchill-Davidson and his colleague Dr. Richardson were the first to observe the existence in man of a dual type of response to neuromuscular blocking agents.

I agree with Dr. Churchill-Davidson that the observations he mentions strongly support his contention that with decamethonium in myasthenia—and probably, by inference, with suxamethonium in normal subjects—the motor endplate alters its response to the blocking agent rather than that the latter is some way alters and consequently elicits a different response from an unchanged motor endplate.

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